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1605 HSD17B6

Figure 1605 HSD17B6 involved in sex hormone metabolism

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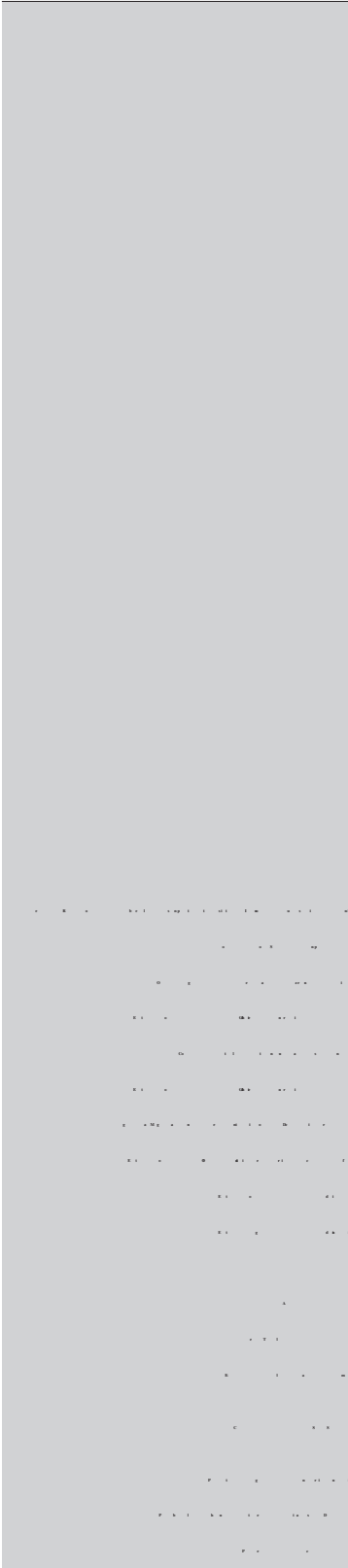
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分子诊断与治疗杂志

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QHF >>MO	.11. .112
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^j QLO	.130
KIOM0	.134
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LMK ^@OM O>KHI I@O E>IM	.143
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COMMENTS

Advances in laboratory diagnostic studies of brucellosis

T P I E T T *

ORIGINAL ARTICLES

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

q q *

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

f f f f f *

Construction of a nomogram model for predicting the risk of pancreatic fistula after pancreatic2M] 0 Mu] MF

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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 β 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 β CAR VEGF IFN γ 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 γ and TSGF levels

The significance of serum IL 1 β 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 α expression and disease activity in patients with ankylosing spondylitis

Efficacy of butylphthaloin combined with Edaravone 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 κ B PCT and IL 1β in cerebrospinal fluid after external ventricular drainage

Effect of Huangqi Yishen Granules combined with sevelamer on TLR4 and NF κ 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 α 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 Xiaocong 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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Hsp90α CER AFP TACE

1 2

90 Hsp90α CER AFP

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2 mRECIST

Hsp90α CER AFP

TACE

ROC TACE TACE TACE

72.86% /

10.928 P<0.05 / TACE

t=2.501 2.082 2.964 P<0.05 TACE

t=7.196 6.866 8.687 P<0.05 / TACE

AFP F=4.966 4.817 7.878 P<0.05

Hsp90α CER AFP

t=8.169 3.434 10.276 P<0.05 / ROC

P<0.05 /

TACE

Hsp90α CER AFP

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 Hsp90α 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 Hsp90α 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 $t/ \chi^2=5.440$ 10.928 $P<0.05$. Before TACE levels of serum Hsp90α 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 Hsp90α 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 Hsp90α 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 α CER and AFP in patients with irregular tumor 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 α combined with CER and AFP before TACE for predicting the curative effect was the greatest $P<0.05$ Serum levels of Hsp90 α CER and AFP can help predict the clinical effectiveness of TACE. These markers can provide a basis for clinical intervention.

Primary liver cancer TACE Hsp90α CER AFP									
transcatheter arterial chemo /									
embolization TACE TACE 2									
immune modified Response Evaluation Criteria In Solid Tumors mRECIST 8 /									
TACE 3 / ≥30%									
TACE /									
90 heat shock protein Hsp90α / - - ≥20%									
Hsp90α - /									
1.3 TACE - 2									
5 mL 10 min 3 500 r/min									
10 cm /									
Hsp90α AFP Roche 2010									
CER /									
CER /									
alpha fetoprotein AFP /									
AFP 6 / Hsp90α CER 1.4									
TACE / SPSS 22.0									
n % 2									
1.1 t t - /									
ROC									
TACE / P<0.05									
2									
2.1 TACE 2 72.86% /									
2.2 70 TACE									
n=51 n=19 /									
P<0.05 / 1 /									
2.3 TACE -									
TACE -									

	AUC	95% CI			
Hsp90α	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	B 6CER	
	0.826		A	■	CER
				ī	1
					Š CER

2

Table 2 Comparison of serum indexes between the two groups

	n	Hsp90α 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	Hsp90α 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		

PMS2 MTA1 VCAM 1

1 VCAM 1		2 PMS2 -	1 MTA1
		/	2019 1 2021 3
81		/	81
4 cm		PMS2 MTA1 VCAM 1	
PMS2 MTA1 VCAM 1		COX	
PMS2 MTA1 VCAM 1		/	PMS2 MTA1 VCAM 1
		P<0.05 / TNM	~ PMS2 + -
MTA1 + VCAM 1 +		~	PMS2 + MTA1 + VCAM 1 +
-			-
PMS2 + MTA1 + VCAM 1 +			-
P<0.05		PMS2 + MTA1 + VCAM 1 +	
P>0.05 / COX		G3 TNM	~ - PMS2 + MTA1 + -
VCAM 1 +		P<0.05 COX	G3 -
TNM		~ -	PMS2 + MTA1 + VCAM 1 +
P<0.05 / PMS2			² =6.688 P<0.05 MTA1
		² =7.161 P<0.05 VCAM 1	
² =9.527 P<0.05 /		PMS2 MTA1 VCAM 1	
		/	
PMS2 MTA1 VCAM 1			

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<

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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

³ /

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<
 P<0.05 / 0.05 COX G3 -
 2 TNM - PMS2 + -
 2.1 PMS2 MTA1 VCAM 1 P<0.05 / 3 /
 PMS2 MTA1 VCAM 1 2.4 PMS2 MTA1 VCAM 1
 P< PMS2 3 81.25% 39/
 0.05 / 1 / 48 54.55% 18/ P<0.
 2.2 PMS2 MTA1 VCAM 1 5 %18/ 5 5 %
 4< >0 <0.
 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	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	1.554	1.081~2.233	0.007
			=0	=1	0.713	0.321~1.076	0.102			
	~	=0	~	=1	2.436	1.680~3.428	<0.001	1.487	1.103~2.234	0.008
		=0		=1	3.308	2.188~5.027	<0.001	1.866	1.112~2.576	0.003
PMS2		=0	+ =1		3.483	2.014~5.147	<0.001	2.147	1.108~3.528	<0.001
MTA1		=0	+ =1		4.112	2.371~6.811	<0.001	2.385	1.117~3.863	<0.001
VCAM 1		=0	+ =1		4.414	1.192~14.376	<0.001	2.554	1.081~3.233	<0.001

3

⁸ / PMS2
/

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: PD; POPF; FRS; PCT; albumin; bilirubin; CT

PCT /									
1.2.3									
		341				POPf		n=26	
		n=84		n=29					
2016	ISGPF	POPf		6	35			14 53.85	
								12 46.15	
POPf		POPf		26	9	/		59.36±8.62	
1.3									
		SPSS 26.0		R 4.1.0				12 46.15	
		-						15 57.69	
								10 38.46	
								13 50.00	
t		n %		2	/	min		362.88±100.06	
Logistic				Back wald		mL		200.14±48.06	
						ASA			
/		AUC						3 11.54	
						cm		23 88.46	
								0.39	
Hosmer Lemeshow									
/		P<0.05		/					
2									
2.1	PD	POPf		FRS		201 374		4	
		POPf		POPf					
-		-		-		cm		1 6.4 7526	
-		-		-		cm		15 57.69 61644; 3.2.5 ,	
-		ASA		-		μmol/L		200 200E	
		P>0.05		-		g/L		14.57.	
FRS		-		-		PCT ng/mL		57.3688±	
-		-		-		95% CI		0.740~0.966	
		PCT				2		/ 32.69 / 100 i 3A	
		P<0.05 /		1 /				231 5 7 / , 0 4 1, 0.57	
2.2	PD	POPf		Logistic		Lemeshow		P=0.793 0.688	
		POPf						/ 3 /	
3									
Logistic				≥0.25 cm		POPf		-	
		FRS		≥5					
		≥98 cm		-		≥240 cm		-	
PD		POPf		PCT					
		POPf		P<0.05 /		2 /		POPf	
2.3	PD	POPf						POPf	
						8 /			
						9 10 /			
-		FRS		-		-		ASA	
-		PCT						11 /	
PD	POPf					Gallery		FRS	
		PD		POPf		-		-	
		ROC		AUC		0.889		POPf	
								12 /	

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	6.833	5.259	1.520~18.186	0.010
	0= 1=	1.473	5.424	4.362	1.251~15.203	0.018
	0=<5 1=≥5	1.570	9.640	4.806	1.515~15.247	0.008
	0=<240 cm 1=≥240 cm	1.426	5.375	4.162	1.164~14.879	0.024
	0=<98 cm 1=≥98 cm	1.583	7.632	4.869	1.608~14.737	0.001
		1.674	7.857	5.333	1.610~17.664	<0.001
PCT		-1.522	10.340	0.218	0.082~0.579	<0.001
		1.620	7.863	5.053	1.571~16.251	<0.001

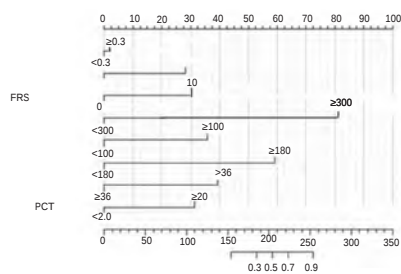


Figure 1 Prediction column line graph model

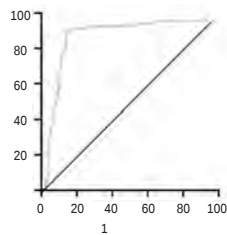


Figure 2 ROC curve of the prediction model in the modeling group

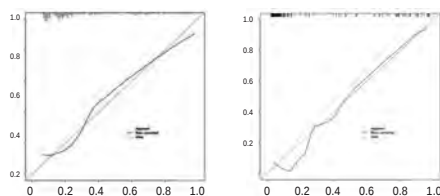


Figure 3 Calibration curves of the predictive model in the modeling and validation groups

Logistic
FRS
PCT
PD
POPF
POPF

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 . 0€

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
	25 59.52	41 55.41	0.185	0.667
	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
>	>
0.05 /	2 /

2.3 MHD	OPN FGF23
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 FGF23 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
	^a P<0.05		^b P<0.05 /

3

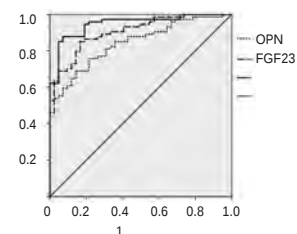
Table 3 Multi factor analysis of vascular calcification in MHD patients

	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 FGF23 levels on vascular calcification in MHD patients

	AUC	95% CI	P
OPN	239.775 0.845	0.777~0.913 0.546 0.857	0.689 <0.001
FGF23	172.135 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

^{10 11} / Fletcher 10UÅ

	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		0.95 100.0	, / 7 U _c
	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 -
G ACI

FT4 -
/ “ r

DAC =μμx 19%50%xA#G' ö`@Öp1
- 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

1 / 24 h 4.5 h /

Free triiodothyronine3 FT3 -

Free thyroxine4 FT4 -

2 / FT3 FT4

3 / 180 /

4 1.2 P>0.05 /

5 / 1.3

ol RC Remnant cholesterol ACI

5 mL 2 000 r/min 10 min

15 cm -80 /

RC FT3 FT4

atherosclerotic

Atellica IM1600

AU5400

FT3 FT4

1 HDL C -

1.1 LDL C - TG -

TC / RC=TC HDL C + LDL C /

2021 1 2023 10

1.4

180 ACI / CT MRI 90 d Rankin mRS 7

1 2018Ne 0~2 mRS /

6 /

1 ACI n %

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

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

/ mRS 3~6 / 90 d
5 3 2 175
116 59 /

1.5

SPSS 22.0 /
— t /
n % ² Logistic /
ACI ROC /
FT3 FT4 RC ACI /
/ P<0.05 /

2

2.1 ACI FT3 FT4 RC
ACI FT3 FT4
RC P<0.05 /
2 /

	n=116	n=59
	54.93±6.24	55.86±6.11
	51/65	23/36
	59/57	26/33
	53/63	32/27
	32/84	43/16
	25/91	50/9
TIA	18/98	10/49
	11/105	6/53
	15/101	8/37
	9/107	1
	6/110	59±0
HDL C mmol/L	1.17±0.36	2.79±0.69
LDL C mmol/L	3.09±0.69	4
TC mmol/L	4.59±0.39	
TG mmol/L	1.62±0.53	
NIHSS	9.31±0.36	
hs CRP mg/L	11.07±2.31	
D D mg/L	3.15	1.85±0.24

3 ±0.73±20.98 41/75
cm 2.79±0.26 5 " i , , " . , 1 5 " . - 1
29/87 51.4. - 1 J Y
/ 1. - 1.5 Ši/ - L Y " .

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

FT3 FT4 RC ACI

AUC 0.951

P<0.05 / 6 1 /

ACI RC

/ ACI

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

/

FT3 FT4 RC

3

ACI

ACI

ACI

/

8 / FT3 FT4

9 / 10

ACI

FT3 FT4

FT3 FT4

1

CD4~+CD25~+Treg

TLR4

hCMV IgM

J .

2024 52 1 123

127.

2

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5

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12 / RC

11 /

13 / RC

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2021 28 1 49 53.
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2020 17 12 734 739+757.
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ProGRP CA242 PCT Hp

				ProGRP CA242 PCT				Hp					
				2020	6	2022	6						
86	/												
			Hp	A		Hp 26	B	Hp 60	/	Hp			
	B			Hp 45				Hp 15	/	A	B	-	
			ProGRP CA242 PCT			ProGRP CA242 PCT			Hp				
			ProGRP CA242 PCT			Hp			/			B	
	ProGRP CA242 PCT			A		t=7.927 8.288 5.419			P<0.05			/	
	ProGRP CA242 PCT					t=11.092 6.897 3.788			P<0.05			/	
	ProGRP CA242 PCT			Hp		r=0.723 0.711 0.688 0.725 0.722 0.701							
	P<0.05 / ROC				AUC	0.912	ProGRP CA242 PCT			0.899			-
	0.871 0.886 P<0.05 /			ProGRP CA242 PCT						Hp			Hp
							Hp			/			
				ProGRP CA242 PCT			Hp						

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To analyze the correlation between serum levels of ProGRP CA242 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 CA242 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 CA242 and PCT levels and Hp infection and prognosis. This study also aimed to evaluate the value of ProGRP CA242 and PCT alone and in combination for detecting Hp infection in intestinal metaplasia of chronic atrophic gastritis. The levels of ProGRP CA242 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 CA242 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 CA242 and PCT levels were positively correlated with HP infection and poor prognosis $r = 0.723 \quad 0.711 \quad 0.688 \quad 0.725 \quad 0.722 \quad 0.701 \quad P < 0.05$. 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 CA242 and PCT alone $P < 0.05$ The levels of ProGRP CA242 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 CA242 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	
^{1 2} /				Hp 45			
Hp	Hp 15	/			25	20	
/	25~81			58.17±5.18	/		
pro gastrin releasing peptide ProGRP	10 A	5 B	-	25~81		58.09±5.21	/
				P>0.05	/		
carbohydrate antigen 242 CA242			3			Hp	
	⁵			RUT -			
/ procalcitonin PCT	3			/ 13C	14C UBT		/
³ /	HPSA		/	Hp			
ProGRP -CA242 PCT				Hp	⁵	13C	14CUPT
Hp		HPSA				-	
1		RUT	/				
1.1	1.2			ProGRP -CA242 -PCT			
2020 6	2022 6			5 mL /			
86				8 cm 3 500 r/min	10~15		
>18 ℓ	Ng ^d	min	/	-		-20	
			/				
ProGRP -CA242 -PCT		Hp	/				
	-		LA2000				
/	18			/			
ProGRP -CA242 -PCT		-		-	/		
Hp			/				
- - -							
/	Hp	A	ProGRP -CA242 -PCT	⁶ /			

1.3

SPSS 22.0
 Spearman ProGRP CA242 PCT
 Hp / ROC
 ProGRP CA242 PCT
 Hp / P<0.05
 /

2

2.1 A B ProGRP CA242 PCT
 B ProGRP CA242 PCT A
 P<0.05 / 1 /

2.2 ProGRP
 CA242 PCT
 ProGRP CA242 PCT
 P<0.05 / 2 /

3

2.3 ProGRP CA242 PCT Hp
 Spearman ProGRP CA242
 PCT Hp CA242 PCT Hp
 P<0.05 / 3 /
 2.4 ProGRP CA242 PCT B ProGRP CA242 PCT
 Hp A ProGRP CA242 PCT
 ROC AUC PCT
 0.912 P<0.05 / 4 1 /
 Hp

AUC 0.912 ProGRP CA242
 PCT 0.899 0.871 0.886 /
 Hp ProGRP CA242 PCT
 Hp /
 ProGRP CA242 PCT
 Hp
 ProGRP CA242 PCT
 9 /
 ProGRP
 10 /
 CA242
 CA242
 11 12 /
 /
 PCT
 13 / ProGRP CA242 PCT
 Hp
 Hp
 ProGRP CA242 PCT
 /
 CA242 14 / Hp
 PCT
 15 16 / Hp
 ProGRP
 / Hp ProGRP
 ProGRP Hp
 /
 ProGRP CA242 PCT
 /
 ProGRP CA242 PCT
 Hp Hp
 Hp /

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NLR PLR

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

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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 were 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 were lower than the antibiotic group and the difference were 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.

Reduning Cefathiamidine Bronchopneumonia Neutrophils Lymphocytes Platelet

		84 / 84		n=42		+		n=42		P>0.05	
pneumonia BP /		broncho		1 /							
		1 /		1.2							
		2 /		1.2.1							
		BP									
3 /		-		20200121		H20030648		≤3			
BP		BP		4 /		4 mL 4~6					
		neutrophil NE -		5 mL 7~8		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				50~100 mg/kg					
		/									
1				20201113		H20103656		250 mL			
1.1						2~3		/			
				+							
		2021 4 2022				0.4~0.6 mL/kg					
2		BP 103									
		BP		20200511		Z20050217		150 mL			
2017		No ⁷		BP		5%		1 /d /			
2~8		1						1 /			
		/		1.2.2				-		-	
		/		1.2.3		NLR PLR		-		1	
		7		4				2 /			
2		6		5 mL		2				GRT 6001	
		1		n %		-					

NE -											
NLR NE/				PLR				/			
/								3 000 rpm			
10 cm 10 min											
-											
BY B10140 Hz 1780						Interleukin 6					
IL 6 -				procalcitonin PCT				/			
1.2.4				advers drug reaction ADR							
-				-				-			
				ADR				/			
1.2.5				7							
				/							
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											
4.086											
P											
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	
+				
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	
+				
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

BP

ADR 9.52% 4.76%

IL 6 PCT
BP

1 IL Y 1PCT

NT proBNP Ang NE

1 2 1

NT proBNP - ICM Ang - HF N

/ 2020 10 2023 2 86 ICM HF

43 / 2 / 2 2 NT proBNP Ang NE

2 / 2 74.42% 93.02%

²=5.460 P<0.05 / 2 NT proBNP Ang NE TNF α IL 6

IL 8 LVTWP PWS TVSS t=5.672 20.016 62% 5.672 20.016 62% 5.672 20.016 62%

AHWJ2021b103

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- 246052

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4BW 4B • 7T 1 XhA + 4F ' 4F ' 4B% 4J%` 4F œiE

crosis factor α TNF α 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	3	6.98	40 93.02
43	17	39.53	15	34.88	11	25.58	32 74.42
							5.460
P							0.019

2.2

2

NT proBNP $\bar{\text{Ang}}$ $\bar{\text{NE}}$

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
t	43	5	010.42±912.55	105.44±17.26	306.22±32.41
	43	4	990.87±998.79	105.96±16.87	305.25±33.33
			0.095	0.141	0.137
	P		0.925	0.888	0.892
	2				
t	43	2	133.95±533.39 ^a	41.43±5.13 ^a	101.26±6.23 ^a
	43	2	799.87±555.23 ^a	69.04±7.45 ^a	187.55±8.09 ^a
			5.672	20.016	55.416
	P		<0.001	<0.001	<0.001

^aP<0.05 /

2.3

2

LVTWP $\bar{\text{PWS}}$ $\bar{\text{IVSS}}$

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

		n	LVTWP	PWS	IVSS
t	P	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
			0.155	1.408	0.629
			0.877	0.163	0.531
2	t	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
			5.940	6.681	5.865
			<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

		n	IL 6	IL 8	TNF α
t	P	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
			0.105	0.156	0.400
			0.917	0.876	0.690
2	t	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
			5.851	5.258	6.742
			<0.001	<0.001	<0.001

^aP<0.05 /

3

ICM HF

⁸ / ⁹

50% /

HF 1

$\bar{\beta}$

¹⁰ /

¹¹ /

¹² /

ICM HF

/

¹³

ICM

HF

- ICM HF
/
14 ICM HF
NT proBNP $\bar{\text{Ang}}$ $\bar{\text{NE}}$
NT proBNP $\bar{\text{Ang}}$ $\bar{\text{NE}}$
- -

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 a regimen alone the GnRH a + 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

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

1.3.3

0 33 ÷ i = E < / TM
×100%
×100%
×100% /

1.3.4

û• p -
/

1.4

SPSS 22.0

§

rhGH⁷ / IVF ET

⁸ / rhGH

⁹ /

rhGH /

¹⁰ /

¹¹ / FSH E₂ LH

/ FSH

E₂

LH

¹² /

FSH E₂ LH

/ rhGH

FSH E₂ rhGH

GnRH GnRH

FSH LH ¹³ / rhGH

FSH LH

rhGH ¹⁴ /

1 IGF + J p /

1 1 1 1 2

TKI / 2020 1 2023 6 CML TKI 80 CML

MMAS 8 TKI /

Cox

TKI - / Cox

MMR -

TKI

P<0.05 /

TKI P<0.05 / /

P<0.05 / Kaplan Meier r

<0.05 /<0.05 /

P<0.05 /

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 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 CML 1.2.2 Chi
² / nese version of the Morisky Medication Adherence
Scale MMAS 8 ⁶ 0~8
³ / TKI <6 ≥6 CML
^{4 5} / CML TKI / 80 100% /

1.2.3 36 36 item short form
health survey SF 36 ⁷ CML - - -
2020 1 2023 6 8 - - - /
CML 80 /
CML 0~100
/ 80 100% /

TKI 1.3 SPSS 22.0 /
No⁵ 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	%
	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
	55	23
		2
		21
P		7.50
		55.00
		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	4.392	1.523	1.028-2.255	0.036
	<70	=1 ≥70	=0	0.782	0.249	0.457	0.274-0.761	0.002
	=1	=0	0.937	0.231	16.451	2.553	1.614-4.038	<0.001
TKI	=1	=0	1.224	0.412	8.855	3.402	1.494-7.750	0.003

4 MMR

Table 4 Comparison of MMR response rates between the two groups

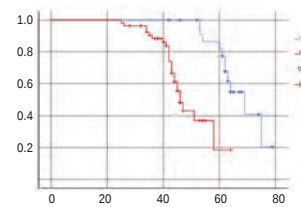
	n	MMR	MMR
	25	22	88.00
	55	30	54.55
		/	7.682
P		/	0.006

62.35±12.39

39.04±10.07

P<0.05 /

1 /



1 KalPan 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

CML

TKI

MMR

PFS TKI 89 /

	-	-			62	38	43.81±5.39
				12 /			21.69±1.43 kg/m ² /
						P>0.05 /	/
			-		1.2		
					1.2.1		
	3 /			/	- - -	- - -	
albumin ALB					-		total
-					bilirubin TBIL	-	glutamic pyruvic
-					transaminase ALT	-	glutamic oxalo
4 /		alkaline phosphatase ALP			acetic transaminase AST	C	C reactive
			-	-	protein CRP	/	
	5 /				1.2.2		
AAPR						126	117
							9
	6 /						-
AAPR			/	/	/		
1					1.2.3		
1.1							5 mL
	2021	11	2023	12	20	4 500 r/min	13.5 cm
			126		15 min		
		59	67	43.52±	/		ALB AAPR
5.14			21.75±1.37 kg/m ²				/
	2.18±0.94	/			1.3		
	7						
		/					
		-	-				
/							
	100						

1.4

IBM SPSS 25.0

—

t

n %

ROC ALB ALP

AAPR

logistic

/ P<0.05

/

2

2.1 ALB ALP AAPR Table

ALB AAPR ALP

P<0.05 / 1 /

1 ALB ALP AAPR —

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 /

2 ALB ALP AAPR —

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 /

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

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		
	43.91±3.81	43.18±3.07	1.013	0.313
	2.06±0.64	2.27±0.39	1.813	0.072
kg/m ²	21.36±0.72	21.14±0.69	1.554	0.123
	39 49.37	14 60.87	0.944	0.331
	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

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	0.0	0.0	0.0
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				

1.3

1.3.1

2 - 1
/ 1 α TNF α -
10 IL 10 - 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
- t /
P<0.05 /

2

2.1

2 1 FVC FEV1 PEF
P<

0.05 / 2 /

2.2

- - - -
P<0.05 / 3 /

2 2

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 /

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
	58	1.91±0.38	3.62±0.55	3.34±0.41	5.31±0.47	7.49±0.23
	58	2.69±0.45	4.35±0.64	4.02±0.65	6.55±0.62	8.10±0.39
t		10.086	6.588	6.739	12.138	10.260
P		<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									
	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	
									6.202	
P									0.013	

2.4

2 /

2.5

1 TNF α IL 10 CRP PCT
P<0.05 / 5 /

2.6

T CD₄⁺ CD₈⁺
CD₄⁺ / CD₈⁺
P<0.05 / 6 /

3

-
P<0.05 / 3 /

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

	n	TNF α ng/L		IL 10 ng/L		CRP mg/L		PCT μg/L	
		1		1		1		1	
t	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
		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

		CD ₄ ⁺ %		CD ₈ ⁺ %		CD ₄ ⁺ /CD ₈ ⁺	
		1		1		1	
t	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
		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 /

11 12 /

1

2

TNF α IL 10 CRP PCT

CD₄⁺ CD₄⁺/CD₈⁺ CD₈⁺

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Cor E NA

NA		Cor E		NA		Cor E	
76		38		38		38	
T ₁		T ₂		T ₃		T ₀	
1 min		3 min		MAP		HR	
SpO ₂		Cor E NA		P<0.05		P>	
0.05 / T ₀ ~T ₃		HR MAP		T2		HR MAP	
P<0.05 / T ₀		T ₃		Cor E NA			
P<0.05 /							

HU Keke WANG Jianshe ZHENG Xin WU Fangfang

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To explore the effect of self-shaping catheter intubation on the levels of cortisol, epinephrine, E and norepinephrine NA in infants with premature closure of cranial suture.

According to the random number table 76 cases of children undergoing craniotomy 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

QNRC2016801

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E-mail: WFf_800330@163.com

	Cortisol	Cor	Norepinephrine	Epinephrine	E
		-		NA	/
1					
1.1					
12			2020	12	2021
					76
				38	
	38	/		4~10	
	7.08±0.85			American so	
society 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		
/		ℓ		4	No ⁵
		≤12			

/ $\times 100\%$ - /

1.3.2

Cormark Lehane

8

/

1.3.3

T_0 - T_1 -

1 min T_2 - 3 min T_3

Mean arterial pressure MAP

60~100 mmHg - Heart rate HR

110~130 /min - Percutaneous ar

terial oxygen saturation SpO_2 92%~
99% ℓ

Ne^9 /

1.3.4 Cor E NA

T_0 T_3 2 mL

$P>0.05$ / 3 /

3 000 r/min

8 r 15 min

cobas E602

Cor 100~

400 ng/L E <480 ng/L NA

200~500 ng/L ℓ

Ne^9 /

1.4

SPSS 26.0

n % ²

-

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

MAP HR SpO_2

T_0 ~ T_3 HR MAP

T2 HR MAP

$P<0.05$ / SpO_2

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 /

- - -

¹⁰ /

/

1 2 3

[illegible]

(1. The 7th Recuperation Department Qingdao Special Servicemen Recuperation Center Of PLA Navy Qingdao Shandong China 266071 2. Sanatorium Zone Office Qingdao Special Servicemen Recuperation Center Of PLA Navy Qingdao Shandong China 266071 3. Emergency Department Qingdao Central Medical Group Qingdao Shandong China 266071)

To explore the changes of serum neuron specific enolase (NSE), interleukin 1 β (IL-1 β),

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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 β 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 β 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 β and 5 HT levels at admission for patients with insomnia and cognitive dysfunction. . . . The levels of NSE and IL 1 β 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 β 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 β 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 β 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 β and decrease in 5 HT.

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

Ne⁸

≥18

Pittsburgh Sleep Quality Index PSQI⁹ >7

3

2 /

/

P>0.05 1 /

3 /

4 /

1.2

NSE IL 1 β 5 HT

ELISA

serum neuron spe

NSE IL 1 β 5 HT /

cific enolase NSE⁵ -

1 β IL 1 β ⁶ 5

5 hydroxytryptamine 5

1.3

1.3.1

PSQI⁹ 90

PSQI

PSQI

IL 1 β 5 HT

/

NSE⁻ 0~21

17~21 -

12~16

7~11 /

1.3.2

1

1.1

2021 8 2023 8

90

0~30

MoCA

Montre

al cognitive assessment MoCA¹⁰ 90

MoCA

MoCA

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
30		25 83.3	5 16.7	25.98±5.37	3.45±0.53
21		18 85.7	3 14.3	26.33±5.26	3.40±0.57
F/		0.400		0.421	0.117
P		0.819		0.658	0.889
				12.91±1.45	13.05±1.59
				13.10±1.32	10 47.6
				0.139	0.920
				0.870	0.631

≤15 } LT MoCA>15 /

90

41

49 - /

1.4 /

SPSS 21.0 /

t

n %

2 NL. Pearson

NSE IL 1β 5 HT MoCA

/ P<0.05 /

2 . NSE -

2		NSE IL 1β 5 HT		-	
Table 2		Comparison of serum levels of NSE IL 1β and		5 HT among the three groups	
n		NSE ng/mL	IL 1β 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	
		^a P<0.05		^b P<0.05 /	
4		NSE IL 1β 5 HT		MoCA	
Table 4		Comparison of serum levels of NSE IL 1β and		5 HT and MoCA score	
n		MoCA	NSE ng/mL	IL 1β 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
49		42 85.7	7 14.3	25.71±5.01	3.37±0.49
F/		0.897		1.678	1.432
P		0.343		0.097	0.156
				11.98±1.33	11.76±1.29
				0.794	2.588
				0.429	0.108

5 NSE IL 1β 5 HT
Table 5 Predictive efficiency of s 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 1β	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

16 /

NSE

NSE

/

IL 1β

−

17 / IL 1β

/ IL 1β

/

IL 1β

18 /

IL 1β

/ 5 HT

−

19 /

5 HT

12 /

/

5 HT

20 /

5 HT

−

13 /

/

14 /

/

5 HT

IL 1β

NSE

5 HT

5 HT

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NSE −

IL 1β

5 HT

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−

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/

/

NSE

IL 1β

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5 HT

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NSE

- ing memory dysfunction in patients with chronic insomnia disorder J . Sleep Med 2023 112 151 158. 11 1 261.
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- 2023 23 8 716 722. 12 - J .
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1 1 2 1

[illegible]

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/m²

CHF / 15 64.03 ± 6.28 $22.91\pm$

CHF 3.44 kg/m² 22 19 / $P>0.05$ /

CHF 1.2

CHF 12 /

CHF 3

CHF 4 /

CHF $2/5$

Mammalian

target of rapamycin mTOR $3/5$ /

CHF mTOR 40 min $3\sim4$ /

CHF 5 / CHF 3 /

CHF 1.3

mTOR / / 5 mL 30 min

1 $3\ 000$ r/min 10 cm

1.1 10 min

2021 1 2023 12

CHF /

CHF 6

New York Heart Association NYHA

6

CHF /

1.4 mTOR mRNA

5 mL Trizol

RNA

cDNA / cDNA

PI3K AKT mTOR mRNA

90 45

1.5

1 2 $n=44$ 3

$n=43$ / 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

Minnesota Living with Heart Failure
Questionnaire MLHFQ

LC3 Beclin 1 Atg9a PI3K AKT
mTOR mRNA
- -
- /

1.6

SPSS 20.0
- t
t / P<0.05 /

2

2.1

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

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	421.38±54.95	226.48±34.51 ^a
	43	53.12±4.91	56.14±4.10 ^a	415.48±57.18	297.15±37.24 ^a
t		0.442	3.676	0.491	9.183
P		0.659	0.001	0.625	<0.001

^aP<0.05 /

3

Table 3 Comparison of serum autophagy markers between the two groups

	n	LC3 ng/mL		Beclin 1 pg/mL		Atg9a ng/mL	
	44	0.85±0.11	1.32±0.11 ^a	18.12±1.52	27.79±2.42 ^a	0.91±0.09	1.62±0.14 ^a
	43	0.89±0.10	1.14±0.13 ^a	17.89±1.77	22.31±1.95 ^a	0.88±0.07	1.21±0.13 ^a
t		1.774	6.978	0.651	11.593	1.733	14.147
P		0.079	<0.001	0.517	<0.001	0.087	<0.001

^aP<0.05 /

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

	n	6MWDF m		MLHFQ	
	44	301.32±14.58	347.61±22.47 ^a	14.77±1.45	7.13±0.69 ^a
	43	304.18±20.76	320.47±21.85 ^a	14.29±1.71	10.24±1.08 ^a
t		0.766	7.814	1.413	16.140
P		0.452	<0.001	0.161	<0.001

^aP<0.05 /

2.3

LC3 Beclin 1 Atg9a
P>0.05

LC3 Beclin 1 Atg9a
P<0.05 / 3 /

2.4

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

2.5

Myo Cr ALT AST
P<0.05 / 5 /

3

CHF

CHF

/

4 PI3K AKT mTOR mRNA -

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

		PI3K		AKT		mTOR	
n							
t	44	0.97±0.10	1.66±0.13 ^a	1.02±0.09	1.78±0.18 ^a	1.04±0.11	1.60±0.13 ^a
	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
		1.399	14.315	1.095	13.383	1.854	8.634
	P	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 $\mu\text{g/mL}$	Cr $\mu\text{mol/L}$	ALT U/L	AST U/L
44	49.58 $\pm$ 5.52	75.68 $\pm$ 8.12	21.38 $\pm$ 4.42	24.47 $\pm$ 3.81
43	50.11 $\pm$ 6.14	77.12 $\pm$ 8.95	21.09 $\pm$ 3.59	25.01 $\pm$ 4.12
	0.424	0.786	0.335	0.635
	0.673	0.434	0.738	0.527

The diagram illustrates a network of interactions between various proteins and pathways. Key nodes and their associated interactions are as follows:

- CHF (Congestive Heart Failure):** A central node with multiple interactions, including:
 - CHF (7, 8) /
 - CHF (9, 10) /
 - CHF (11, 12) /
 - CHF (13) /
 - CHF (14) /
 - CHF (15) /
 - CHF (16) /
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- mTOR:** Interacts with CHF (1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100).
- PI3K/AKT:** Interacts with mTOR (1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100).
- Beclin 1:** Interacts with mTOR (1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100).
- Atg9a:** Interacts with Beclin 1 (1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100).
- LVEF (Left Ventricular Ejection Fraction):** Interacts with CHF (1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100).
- 6MWD (6-Minute Walk Distance):** Interacts with CHF (1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100).
- NT proBNP (Natriuretic Peptide B-type):** Interacts with CHF (1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84,

NLRP3

		DN		NLRP3		DN		80	
/		2022	1	2024	1				
						40		/	
						DN			
						3		/	
3						PBMCs		NLRP3	
		NLRP3							
/		3		FBG		HbA1c			
		t=0.698		0.894		P>0.05			
ACR		PBMCs		NLRP3		Caspase 1		mRNA	
Gasdermin D		N		GSDMD		N			
5.355		7.336		6.654		7.835		P<0.05	
						/		3	
						2=0.581		P>0.05	
						/			
		NLRP3				DN			
						/			
		NLRP3							

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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 β IL 1 β 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		DN		DN	
3		69.23 $\pm$ 7.19		22		18		10.61 $\pm$ 2.85	
DN		3.71 $\pm$ 0.95		21		19		69.18 $\pm$ 6.85	
10.47 $\pm$ 2.92		DN		3.65 $\pm$ 0.92		/		P>	
2		1 glucagon like peptide 1 GLP 1		0.05 /		/		/	
45 /		1.2		DN		DN		/	
DN		DN		/		/		/	
DN		1.5 mL		1.34		0.25 mg		1.34	
1		mg/mL		1		4		0.5 mg	
1.1		1.3		/		3		/	
1.3.1		1.3.1		3		fasting blood glucose FBG		-	
2022 1		2024 1		DN 80		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		-	
-		-		5.0 mL		141		-	

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
 / β 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 β mRNA P>0.05 /
 interleukin 1 β IL 1 β - 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
 - - -
 - - / IL 1 β IL 18 GSDMD N
 1.4 P>0.05 / 3
 SPSS 22.0 / IL 1 β IL 18 GSDMD N
 - P<0.05 /
 t / n % 4 /
 / 2.6
 U / P<0.05 / 3 2
 1 - 1 -
 2
 2.1 3 1 - 2
 -
 FBG HbA1c
 P>0.05 / 3 /
 FBG HbA1c $r^2=0.482$ P>0.05 /

1
 Table 1 Comparison of blood glucose levels between the two groups -

	n	FBG mmol/L		t	P	HbA1c %		t	P
		3				3			
t	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
		0.075	0.698			0.189	0.894		
P		0.941	0.487			0.850	0.374		

2
 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			
	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		
			3				3		
t		40	0.84±0.13	0.76±0.09	3.200	0.002	0.76±0.09	0.66±0.08	5.252
		40	0.82±0.15	0.69±0.08	4.836	0.000	0.74±0.10	0.57±0.07	8.808
			0.637	3.677			0.940	5.355	
P			0.526	0.000			0.350	0.000	

4 NLRP3 pmol/L													
Table 4 Comparison of NLRP3 downstream cytokines in serum between the two groups pmol/L													
	n	IL 1β		t	P	IL 18		t	P	GSDMD N		t	P
		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 I b e

min 10 cm 15 min / P>0.05 T_1 T_2 T_3 IL 6
IL 6 T_L 2 IL 2
/ P<0.05 / 2 /

$CD4^+$ $CD8^+$ $CD4^+/CD8^+$ / $CD3^+$ T 1 min
1.3.3 - - -

/
1.4

SPSS 21.0

- t

t n %

2

P<0.05

/

2

2.1

-

-

2.3

T

T_0 $CD3^+$ $CD4^+$ $CD8^+$ $CD4^+/CD8^+$

P>0.05 T_1 T_2 T_3

$CD3^+$ $CD4^+$ $CD4^+/CD8^+$

$CD8^+$

P<0.05 /

3 /

P<0.05 / 1 /

2.4

2.2

IL 6 T_L 2

T_0 IL 6 T_L 2

P>0.05 / 4 /

2

IL 6 IL 2

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

T_0 ^aP<0.05 T_1 ^bP<0.05 T_2 ^cP<0.05 /

3

T

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 ^{ab}	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 ^{ab}	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

T_0 ^aP<0.05 T_1 ^bP<0.05 T_2 ^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											
										0.164	
P											0.685

3

LC /
79 / -

B C C l - = = ' 2 = ' > = 1

> 7 > > B = -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.

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

Peri implantitis PI		1.2	
		1.2.1	
		2 mm×10 mm	
¹ /		/	
		1 min /	
² / C		C reac	
tive protein CRP			
		/	
		200 μ L	
³ /		Osteopontin	
OPN		1 min	
OPN		3 500 /min	
PI		8 cm 5 min	
		-80 /	
		1.2.2 OPN CRP RANKL	
kB		OPN	
Receptor activator of NF- κ B ligand RANKL		CRP RANKL	
⁵ /		/	
OPN CRP RANKL		PI	
		/	
		1.2.3	
1		2	
1.1		Acute physiology and chronic	
		health evaluation	
		APACHE	
2021 6		⁷ APACHE	
87		60 -	
2023 3		71	
PI		5 -	
50		6	
37		>21	
44.13±5.33		≤21 /	
11		34	
87		42	
		1.3	
80		46	
34		/	
44.59±5.02		0	
10		1	
30		2	
40		3	
80		4	
		/	
PI		PI	
		6	
		-	
		-	
		8 /	
		-	
		30 s	
		0	

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 / PI

13 PI

/ 14 PI
PI PI
PI

/

CRP OPN RANKL

CRP OPN RANKL

PI / OPN

CRP

RANKL

•

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 /

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	85.04

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 /

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

	n=78	n=24	t/	P
	53 67.95	10 41.67	5.368	0.021
	25 32.05	14 58.33		
<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	0.122	0.727
	66 84.62	21 87.50		
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

		LCR			AUC 0.904	LCR	-
	/	LCR		HALP			
CRP	/			/		LCR HALP	
	/						
				/			
					LCR	HALP	
		/ CRP					
		CRP				/	
		¹⁰ / CRP					
				1			
					ACE2 Ang Ang 1 7		J .
					2021 13 12 2008 2012.		
				2			
			^{11 12} /				
	LCR				J .	2022 19 10 46 49.	
		/		3	Li X Peng S		
	HALP		J		2022 209		
		-					
	/		HALP				
			/				
	/	-					
	/						
			¹³ /				
		¹⁴ /					
				/			
	-						
		¹⁵ /		-			
		¹⁶ /	HALP				
-	-	-					
	/						
		LCR HALP					
		AUC 0.837 0.759					

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

68 52
60~75 66.84±7.14 /
 $P>0.05$ /
12 /
1.2
1.2.1 sTREM 1 AQP5
3
Soluble triggering receptor expressed on human myeloid cells 1 sTREM 1 5 Aquaporin 5 mL
5 mL 3 000 r/min
10 cm 10 min -70
34 / sTREM 1
AQP5
1.2.2
2017 №6
n=31 n=115 /
1.3
SPSS 25.0
t
1.1
2021 10 2023 10
146
5
3 d /
87 59 61~79 / 146
67.61±7.69 8~17
9.28±0.94 / 120
2
2.1 sTREM 1 AQP5
sTREM 1 AQP5
 $P<0.05$ / 1 /
ROC
sTREM 1 AQP5
/ $P<0.05$

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

• •

HPV TCT

1	2	1	1	1	1	1	1	1
				HPV	TCT			
/		2022	7	2023	9	10 124		
HPV	TCT	/		10 124	HPV	7.34%	HPV	
² =29.721 P<0.001				/	HPV	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	?	"•	?	"•
				ADLP		s»@ 1 0Q		

				Humanpapillomavirus		HPV DNA		HPV31 HPV52 HPV82	HPV33 HPV56 HPV82	HPV35 HPV58 15	HPV39 HPV59 /	HPV45 HPV66 /	HPV51 HPV68
				200	/								
				HPV									
				HPV				1.2.3					
				¹ /									95%
				2020					/	TBS The bethesda system			
60.4				34.2	/				⁴				
						² /		Negative for intraepithelial lesion or malignancy					
2020					10.97			NILM				Atypical squamous cells	
5.91						18%		ASC				Low grade squamous	
				/	HPV			intraepithelial lesion LSIL					
				Thinprep cytologic test TCT				High grade squamous intraepithelial lesion HSIL	/				
				³ /				1.3					
2022	7		2023	9					SPSS 22.0				
	HPV				TCT				n %		²	Fisher	
										P<0.05			
	/							/					
1								2					
1.1								2.1 HPV					
		2022	7		2023	9					10 124	HPV	
								7.34% / 21~	HPV			41~	
	HPV									HPV			
								10		P<0.001 /			
124			21~87	/			21~				41~		
738		31~ 3	723		41~ 3	090	51~ 1		88.1% 163/185	≥61			
803		≥61 770		/					24.68% 19/77	/			
1.2										P=0.092 /		1 /	
1.2.1								2.2	HPV				
									/ HPV 51 HPV 52	HPV 59			
					5								P<
								0.005 /	2 /				
		4		/				2.3		TCT			
1.2.2	HPV							TCT		2.49% 21~			≥61
									/		TCT		
							HPV			P>0.05 /	3 /		
		PCR						2.4 TCT		HPV			
					HPV16 HPV18					TCT HPV			

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

	n=10 124	21~ n=738	31~ n=3 723	41~ n=3 090	51~ n=1 803	≥61 n=770		P
	9 381 92.66	658 89.16	3 451 92.69	2 905 94.01	1 674 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=10 124	21~ n=738	31~ n=3 723	41~ n=3 090	51~ n=1 803	≥61 n=770		P
NILM	9 872 97.51	712 96.48	3 625 97.37	3 020 97.73	1 762 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% 147 3
1.45% / ASC
LSIL HSIL HPV 51.41% HPV
70.18% 88.89% HPV
P<0.001 /
HSIL 37.5% 6/16 HPV
LSIL 77.5% 31/40 / DNA HPV E2
HPV E6 E7
P<0.001 / 4 / DNA
TCT
1 3 /
/ HPV 16 HPV 45 HPV TCT
66 P<0.005 / HPV
5 /

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

n=10 124	NILM n=9 872	ASC n=177	LSIL n=57	HSIL n=18	P
9 381 92.66	9 276 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

10 124 HPV /
7.43% 5 TCT 2.49% 7
6 9 HPV 6 8 12 / ASC HSIL
/ 12 / LSIL
21~ 61 HPV 21~ 6
31~ 41~ HPV / HPV
ñ UÑ 6 7 / LSIL HPV 70.18% HSIL
88.89% / LSIL HSIL
15 - 6
/ 12 LSIL HSIL
HPV HPV / TCT NILM ASC HPV
52 HPV 58 HPV 51 HPV 16 HPV 39 / HPV 52 HPV 51 LSIL
10 11 HPV 66 HPV 52 HPV 56 HSIL 16 52 /
52 HPV 58 HPV 16 HPV
5 HPV 12 /
/ HPV HPV
/ HPV
HPV HPV /
HPV 52 HPV 58 HPV 16 HPV 51 13 /
2014 2018 14 HPV
/ 1

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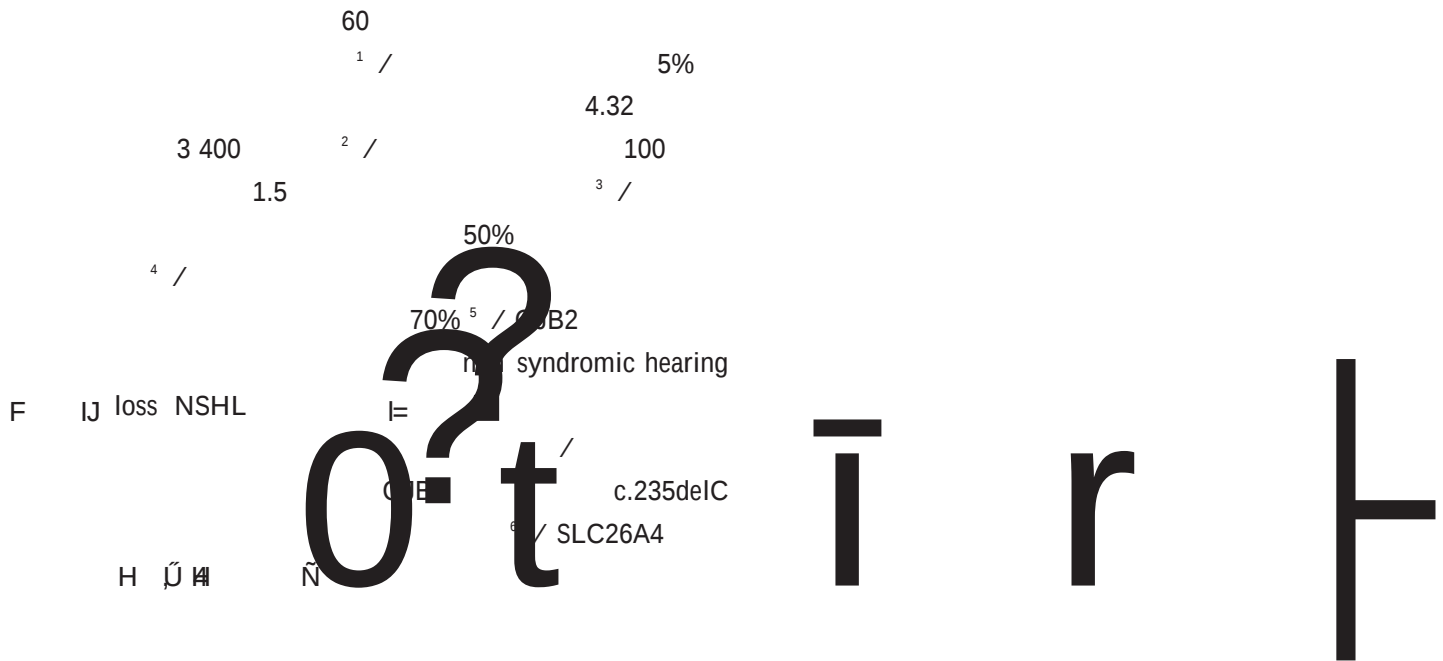
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c.235delC showed significant differences between the two nationalities. The GJB2 c.235delC and SLC26A4 c.919 2A >G mutations were most commonly found in patients with NSHL in our region. The frequency of the GJB2 gene mutation in Han patients was significantly 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		/
						56.32%	49/87
2.1					GJB2	29.89%	26/87
	62	49.6%			SLC26A4	22.99%	20/87
		31	24.8%			34.21%	13/38
GJB2		26	20.8%		GJB2	13.16%	5/38
SLC26A4	3	2.4%		GJB3	SLC26A4	15.79%	6/38 /
2	1.6%		MT CO1		2 /		

Table 2 Genetic mutations of NSHL patients in the Inner Mongolia region 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
	c.235delC	6 6.90	1 2.63
SLA26A4	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				
25.29%	55/250	SLC26A4	15.6%	39/250
GJB3	1.2%	3/250		
MT CO1	1.6%	4/250		/

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

2.2				
		GJB2		GJB2
c.235delC				
P<0.05 /		4 /		

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

		n=174	n=76
GJB2	c.235delC	35 20.11	6 7.89
	c.299_300delAT	7 4.02	2 2.63
	c.176_191del16	1 0.57	1 1.32
	c.35delG	1 0.57	0 0.00
	c.427C>T	1 0.57	0 0.00
	c.512insAACG	1 0.57	0 0.00
SLC26A4	c.919 2A>G	17 9.77	6 7.89
	c.2168A>G	6 3.45	2 2.63
	c.1336C>T	3 1.72	0 0.00
	c.281C>T	2 1.15	0 0.00
	c.2027T>A	1 0.57	0 0.00
	c.1174A>T	1 0.57	0 0.00
	c.1181_1183delTC	1 0.57	0 0.00
GJB3	c.538C>T	2 1.15	1 1.32
MT CO1	m.7444G>A	2 1.15	2 2.63

TCCD ONSD APACHE

TCCD					ONSD				
APACHE					/				
TCCD					ONSD				
2023 1					2023 12				
n=100					n=100				
/					/				
CK					WBC				
ALT					HGB				
PT					TBIL				
SOFA					APACHE				
GCS					1				
APACHE					SOFA				
SOFA					GCS				
P>0.05					P<0.05				
SOFA					TCCD				
GCS					ONSD				
2=5.307 P<0.05 /					/				

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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

Study	Study Design	Study Period	Study Location	Study Population	Study Interventions	Study Outcomes	Study Limitations
1	Retrospective	2023	100	100	Transcranial color Doppler ultrasound	55.72±8.37	1.2
2	Retrospective	2023	100	100	Transcranial color Doppler ultrasound	55.48±	1.2
3	Retrospective	2023	100	100	Transcranial color Doppler ultrasound	55.48±	1.2
4	Retrospective	2023	100	100	Transcranial color Doppler ultrasound	55.48±	1.2
5	Retrospective	2023	100	100	Transcranial color Doppler ultrasound	55.48±	1.2
6	Retrospective	2023	100	100	Transcranial color Doppler ultrasound	55.48±	1.2

Table 1 Comparison of blood indexes in patients with cerebral resuscitation after cardiac arrest by different monitoring methods1 ^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	
P								5.307	
								0.021	

3

-

	/	6		2019 J .
				2019 11 1034 1041.
		7		
	/		IL 6 Ghrelin NSE	J .
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ONSD	/	8	Meidert AS Buschmann D Brandes F et al. Molecular RNA Correlates of the SOFA Score in Patients with Sepsis	
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TCCD	/	9	r	-
ONSD	-	-	-	-
	/			

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Tuberculosis Spleen invigorating lung pill β CAR VEGF IFN γ IL 17[illegible]

β CAR $\bar{\text{VEGF}}$ $\bar{\text{IFN}} \gamma$ $\bar{\text{IL}} 17$

/

1.4.4

90 d

CytoFLEX

CD3⁺ -CD4⁺ -CD8⁺

CD4⁺/CD8⁺

1.4.3 /

1.4.5

90 d

CHESTAC 8800

Forced expiratory volume FEV1 -

forced vital capacity FVC $\overline{\text{FEV1}}/\text{FVC}$ /

1.5

SPSS 23.0

U•

c v n g 1 T

1

	n	%
1. <i>How often do you use the Internet?</i>		
a. Every day	10	100
b. Several times a week	10	100
c. Once a week	10	100
d. Several times a month	10	100
e. Once a month	10	100
f. Several times a year	10	100
g. Once a year	10	100
h. Never	10	100
2. <i>How often do you use the Internet for work?</i>		
a. Every day	10	100
b. Several times a week	10	100
c. Once a week	10	100
d. Several times a month	10	100
e. Once a month	10	100
f. Several times a year	10	100
g. Once a year	10	100
h. Never	10	100
3. <i>How often do you use the Internet for shopping?</i>		
a. Every day	10	100
b. Several times a week	10	100
c. Once a week	10	100
d. Several times a month	10	100
e. Once a month	10	100
f. Several times a year	10	100
g. Once a year	10	100
h. Never	10	100
4. <i>How often do you use the Internet for entertainment?</i>		
a. Every day	10	100
b. Several times a week	10	100
c. Once a week	10	100
d. Several times a month	10	100
e. Once a month	10	100
f. Several times a year	10	100
g. Once a year	10	100
h. Never	10	100
5. <i>How often do you use the Internet for social networking?</i>		
a. Every day	10	100
b. Several times a week	10	100
c. Once a week	10	100
d. Several times a month	10	100
e. Once a month	10	100
f. Several times a year	10	100
g. Once a year	10	100
h. Never	10	100
6. <i>How often do you use the Internet for news?</i>		
a. Every day	10	100
b. Several times a week	10	100
c. Once a week	10	100
d. Several times a month	10	100
e. Once a month	10	100
f. Several times a year	10	100
g. Once a year	10	100
h. Never	10	100
7. <i>How often do you use the Internet for education?</i>		
a. Every day	10	100
b. Several times a week	10	100
c. Once a week	10	100
d. Several times a month	10	100
e. Once a month	10	100
f. Several times a year	10	100
g. Once a year	10	100
h. Never	10	100
8. <i>How often do you use the Internet for business?</i>		
a. Every day	10	100
b. Several times a week	10	100
c. Once a week	10	100
d. Several times a month	10	100
e. Once a month	10	100
f. Several times a year	10	100
g. Once a year	10	100
h. Never	10	100
9. <i>How often do you use the Internet for research?</i>		
a. Every day	10	100
b. Several times a week	10	100
c. Once a week	10	100
d. Several times a month	10	100
e. Once a month	10	100
f. Several times a year	10	100
g. Once a year	10	100
h. Never	10	100
10. <i>How often do you use the Internet for travel?</i>		
a. Every day	10	100
b. Several times a week	10	100
c. Once a week	10	100
d. Several times a month	10	100
e. Once a month	10	100
f. Several times a year	10	100
g. Once a year	10	100
h. Never	10	100

Table 1 Comparison of clinical efficacy between the two groups

[illegible]

2

d

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

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

3

Table 3 Comparison of serum cytokine levels between the two groups

		n	β CAR μmol/L	VEGF pg/mL	IFN γ pg/mL	IL 17 pg/mL
90 d	t	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
			0.717	0.406	1.634	0.400
		P	0.475	0.685	0.105	0.690
	t	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
			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

IL 17 IFN γ

90 d β CAR VEGF IL 17
 CD8⁺ IFN γ
 CD3⁺ CD4⁺ CD4⁺/CD8⁺ FEV1 FVC FEV1/
 FVC

T
 β CAR VEGF IFN γ IL 17

A

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 electioe leve aes sbecv 0.05dneels leñ

-80 /
CD34+ / <2×10⁶/kg
CD34+ / ≥2×10⁶/kg CD34+
≥ 5 ×10⁶/kg /
1.2.3
CD34+
Beackman navios /
1.2.4
- - BMI -
- CD34 + - -
/
NIH CTCAE 4.0 /
1.3
SPSS 22
/ qt (R=0.658) *T L†T=0.68HbX g %S
"À%S

1

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

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

2

Table 2 Univariate analysis affecting stem cell collection

	n	%	t/	P
	n=25	n=13		
	16	64.00	6	46.15
	9	36.00	7	53.85
			6.545	0.015
	18	72.00	6	46.15
	7	28.00	7	53.85
			2.455	0.117
	12	48.00	5	38.46
	13	52.00	8	61.54
			2.040	0.199
	11	44.00	7	53.85
	15	56.00	6	46.15
			2.001	0.203
	19	64.00	5	38.46
	6	36.00	8	61.54
			13.25	<0.01
	14.02±18.55	5.96±7.38	1.901	0.054
	4.81±1.25	4.75±1.07	0.158	0.896
	1.51±0.81	1.45±0.88	0.196	0.877
	1.18±0.31	1.14±0.25	0.354	0.775
	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	-2.786	1.265	4.852	0.062	0.005~0.736	0.028
LDL C	<	=1	LDH C≥	2.95	1.199	6.055	19.109	1.823~200.329	0.014

2.4

^{11 12} /

2019

CD34+

G CSF

 $R^2=0.4128$ P<0.01 /¹¹ /

2.5

MM

G CSF

MM

/ 4 /

4

%

Table 4 Adverse 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

3

MM

CD34+

auto HSCT

MM

⁷ /

/

G CSF

⁸ /⁹

1 SDF 1

/

SDF 1

4

CXCR4

CXCR4

^{13 15} /¹⁰ /

G CSF

CXCR4

-

1 SDF 1

CXCR4

SDF 1

CX

CR4

SDF 1

/

PCT SAA Treg

PCT SAA Treg

380

378 / PCT SAA Treg

IL-6 / Logistic PCT SAA Treg IL-6 PCT SAA Treg IL-6

P<0.05 / PCT SAA Treg IL-6

Logistic PCT >0.5 µg/L SAA >10 mg/L

TGF β IL-6 PCT SAA Treg IL-6 P>0.05 7 d PCT

SAA IL-6 TGF β P<0.05 / ROC PCT

SAA TGF β IL-6 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 as 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 β (TGF β) and interleukin 6 (IL-6) 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 β and IL-6 levels of the patients in the test group with different efficacies before and after the treatment. The levels of PCT, SAA, TGF β and IL-6 individually and in parallel to assess the value of antibiotic treatment efficacy were analyzed. The

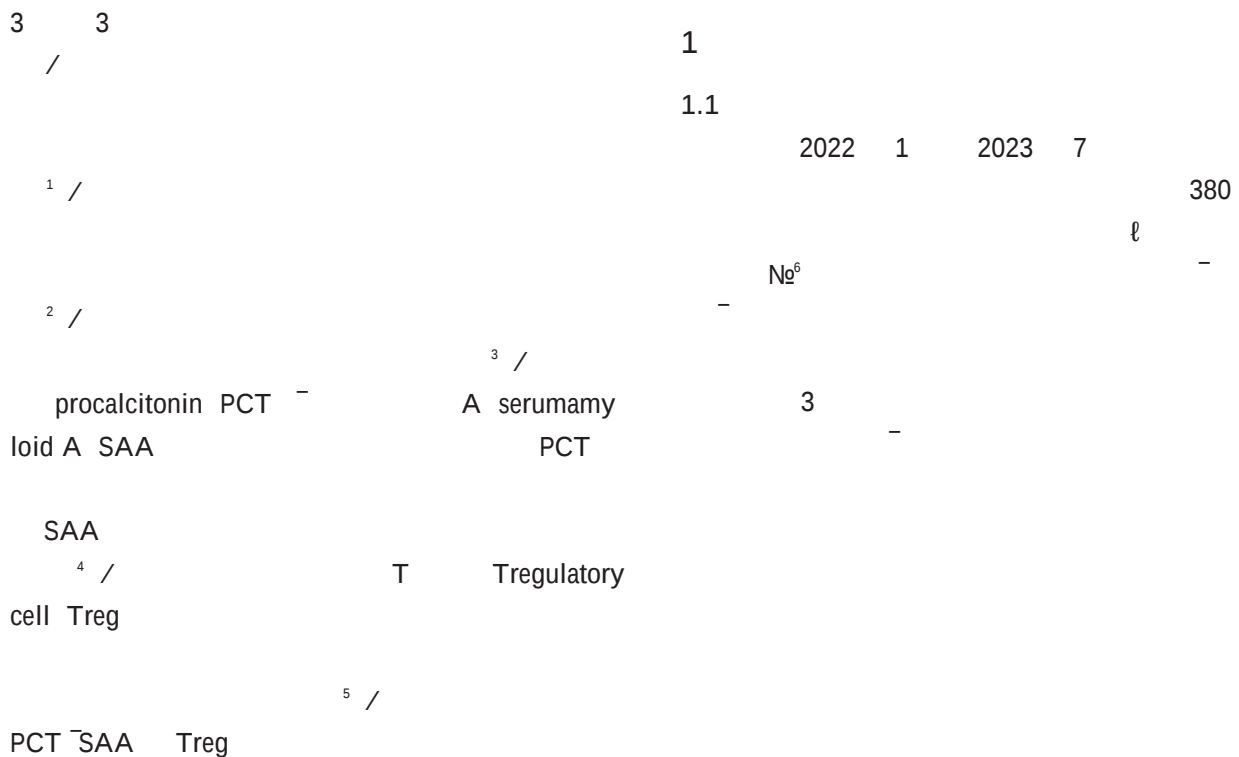
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serum PCT SAA and ILA7A levels in the test group were higher than those in the control group and the TGF β level was lower than that in the control group and the difference was statistically significant $P<0.05$. Univariate regression analysis showed that serum PCT SAA TGF β and ILA7A 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 β and elevated ILA7A 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 β and ILA7A between the two groups before treatment $P>0.05$ and the levels of PCT SAA and ILA7A in the effective group were lower than those in the ineffective group and the level of TGF β 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 β and ILA7A 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	T ₁	^	.U<
	378	0.37±0.04	7.16±2.26	0.02	5.31±0.02	1.1±2.69
	380	4.69±0.48	118.55±28.43			6 48.001<
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/=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 β	X ₃		1.596	1.223~2.084	0.015	1.736	1.137~2.652	<0.001
ILA7A	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	Gensini
sLOX 1 sST2 SREBP 1	QRS		sLOX 1 sST2 SREBP 1
QRS	Spesrman	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

2023WZ12

1. 010017

2. 010017

E mail nmgrmyy2005ydy@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.

		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	
		oxidized low density lipoprotein ox LDL		toris SAP 51		Unstable angina	
		CHD		pectoris UAP 63		acute myocar	
sLOX 1		LOX 1		dial infarction AMI 42 /			
/		1 Sterol regulatory element binding protein 1 SREBP 1		CHD 63 /		52	
		17p11.2		41 /		50	
		/		P>0.05 1 /			
		2				/	
soluble growth STimulation expressed gene2		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		/	
sST2		/		sLOX 1 sST2		ELISA	
				1.2.2 SREBP 1		R&D /	
1						5 mL	
1.1				12 cm 3 500 r/min		10 min	
				TRIZOL		RNA	
2020 4 2022 4				Takara Japan		1 μ g RNA	
CHD 156 CHD				cDNA cDNA		PCR	
CHD ³				/		F TGCATTTTCTGACACGCTTC	

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

	n										kg/m ²		
	50	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

AMI >UAP >SAP >

95 30 s 95 10 s 60 35 s 70 60 s

P<0.05 / 2 /

40 / SREBP 1 2^{ΔΔt} /

2 CHD sLOX 1 sST2 SREBP 1 QRS

1.2.3 Gensini

CHD

0

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 /

1.3

SPSS 20.0

n %² Pearson

sLOX 1 SREBP 1 sST2 Gensini

QRS Spesrman

sLOX 1 SREBP 1 sST2

P<0.05 /

2

2.1 CHD sLOX 1 sST2 SREBP 1

QRS

sLOX 1 sST2 SREBP 1 QRS

3 Gensini sLOX 1 sST2 SREBP 1 QRS

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 /

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

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

2.3 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

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	
	^a P<0.05	^b P<0.05 /				

Table 5 Correlation between changes in sLOX 1 sST2 and SREBP 1 and Gensini score number of coronary lesions

	Gensini				QRS	
sLOX 1	0.439	<0.001	0.539	<0.001	0.474	<0.001
	0.556	<0.001	0.663	<0.001	0.592	<0.001
sST2	0.491	<0.001	0.618	<0.001	0.501	<0.001

	CHD	sLOX 1	SREBP 1	sST2	8	. sST2	NLR	
						J .		2023 15
	CHD	/			10 1685 1688 1693.			
					9	.		1 ⁻
						1		
						J .	2020 41 1	101 103.
1	Shaya GE Leucker TM Jones SR et al. Coronary heart disease risk Low density lipoprotein and beyond J . Trends Cardiovasc Med 2022 32 4 181 194.				10	1	A20	J .
							2021 23 5	475 478.
2	Cruz DE Tahir UA Hu J et al. Metabolomic analysis of coronary heart disease in an african american cohort from the jackson heart study J . JAMA Cardiol 2022 7 2 184 194.				11	.	sST2 NLR	
						J .		2021 31
3	. .7 M .				12	1 38 41.		
	2010 203 210.					Sheikh MSA. Role of plasma soluble lectin like oxidized low density lipoprotein receptor 1 and microrna 98 in severity and risk of coronary artery disease J . Balkan Med J 2021 38		
4	.			J .		1 13 22.		
	2007 35 3 195 206.				13	.	AQP9 ⁻ HIF 1 α	
5	. GGT						J .	
						2023 20 7 27 30.		
	J .			2021 20 15 1603	14	.	sST2	
	1607.					QRS	J .	
6	.					2020 60 33 79 82.		
	1			J .	15	. sST2 Treg		
				2022 20 3 515 519.		J .	2018 18 7 913 918.	
7					16	.		
	1					SREBP 1 mRNA		
	J .			2020 20 6 605 607.		J .	2021 36 5 1 7.	

1514

3	.			Treg	10	.		
	J .					J .		2022
				2022 14 7 1237 1241.		17 1 114 117.		
4	.				11	.		
	J .			2022 23 3 154 157.		2022 21 5 600 604.		
5	.			c di AMP	12	.		
	J .			2022 53 6 1098 1103.		SAA CRP		J .
6	.			J .		2023 18 8 961 964.		
	2001 81 5 314 320.				13	. PCT ⁻ Th17 Tregs ACLF		
7	.			PCT ⁻ SAA IL 6		J .		2022 14 8
						1350 1353.		
	J .			2021 34 12 45 48 63.	14	Dorresteijn KRIS Brouwer MC Korné Jellema et al. Bacte rial external ventricular catheter associated infection J . Ex pert Review of Anti infective Therapy 2020 18 5 1 11.		
8	. ℓ			2018 Ne J .		. ZEB1	NNMT	
	2021 56 9 951 953.				15	J .		
9	. ADA ⁻ PGLYRP2 PCT							
	J .			2023 20 4 42 45.		2021 18 5 360 365.		

IL 6 CRP Hcy ACA

ACA	146	6 IL 6	CRP	Hcy
	n=109	n=37	2020 9 2023 9	Rankin mRS
NIHSS				
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

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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

1.2		1.2.1		1.2.2	
6	interleukin 6 IL-6	BMI	body mass Index	NIHSS	
2	/ C	CRP	C reactive protein	15	42
3	/	Hcy	homocysteine	84	62
4		lipin antibody	ACA	91	55
5	/	IL-6	CRP	91	55
6		IL-6	CRP	91	55
7		IL-6	CRP	91	55
8		IL-6	CRP	91	55
9		IL-6	CRP	91	55
10		IL-6	CRP	91	55
11		IL-6	CRP	91	55
12		IL-6	CRP	91	55
13		IL-6	CRP	91	55
14		IL-6	CRP	91	55
15		IL-6	CRP	91	55
16		IL-6	CRP	91	55
17		IL-6	CRP	91	55
18		IL-6	CRP	91	55
19		IL-6	CRP	91	55
20		IL-6	CRP	91	55
21		IL-6	CRP	91	55
22		IL-6	CRP	91	55
23		IL-6	CRP	91	55
24		IL-6	CRP	91	55
25		IL-6	CRP	91	55
26		IL-6	CRP	91	55
27		IL-6	CRP	91	55
28		IL-6	CRP	91	55
29		IL-6	CRP	91	55
30		IL-6	CRP	91	55
31		IL-6	CRP	91	55
32		IL-6	CRP	91	55
33		IL-6	CRP	91	55
34		IL-6	CRP	91	55
35		IL-6	CRP	91	55
36		IL-6	CRP	91	55
37		IL-6	CRP	91	55
38		IL-6	CRP	91	55
39		IL-6	CRP	91	55
40		IL-6	CRP	91	55
41		IL-6	CRP	91	55
42		IL-6	CRP	91	55
43		IL-6	CRP	91	55
44		IL-6	CRP	91	55
45		IL-6	CRP	91	55
46		IL-6	CRP	91	55
47		IL-6	CRP	91	55
48		IL-6	CRP	91	55
49		IL-6	CRP	91	55
50		IL-6	CRP	91	55
51		IL-6	CRP	91	55
52		IL-6	CRP	91	55
53		IL-6	CRP	91	55
54		IL-6	CRP	91	55
55		IL-6	CRP	91	55
56		IL-6	CRP	91	55
57		IL-6	CRP	91	55
58		IL-6	CRP	91	55
59		IL-6	CRP	91	55
60		IL-6	CRP	91	55
61		IL-6	CRP	91	55
62		IL-6	CRP	91	55
63		IL-6	CRP	91	55
64		IL-6	CRP	91	55
65		IL-6	CRP	91	55
66		IL-6	CRP	91	55
67		IL-6	CRP	91	55
68		IL-6	CRP	91	55
69		IL-6	CRP	91	55
70		IL-6	CRP	91	55
71		IL-6	CRP	91	55
72		IL-6	CRP	91	55
73		IL-6	CRP	91	55
74		IL-6	CRP	91	55
75		IL-6	CRP	91	55
76		IL-6	CRP	91	55
77		IL-6	CRP	91	55
78		IL-6	CRP	91	55
79		IL-6	CRP	91	55
80		IL-6	CRP	91	55
81		IL-6	CRP	91	55
82		IL-6	CRP	91	55
83		IL-6	CRP	91	55
84		IL-6	CRP	91	55
85		IL-6	CRP	91	55
86		IL-6	CRP	91	55
87		IL-6	CRP	91	55
88		IL-6	CRP	91	55
89		IL-6	CRP	91	55
90		IL-6	CRP	91	55
91		IL-6	CRP	91	55
92		IL-6	CRP	91	55
93		IL-6	CRP	91	55
94		IL-6	CRP	91	55
95		IL-6	CRP	91	55
96		IL-6	CRP	91	55
97		IL-6	CRP	91	55
98		IL-6	CRP	91	55
99		IL-6	CRP	91	55
100		IL-6	CRP	91	55

n %	2	Logistic	3
		Pearson	Table 3
	mRS		Multivariate analysis of prognosis in patients with acute cerebral infarction
	ROC		
/	P<0.05	/	
2			
2.1		P>0.05	
	NIHSS		
	P<0.05	/	1 /
1	n %	-	
Table 1 Comparison of general data between 2 groups			
	n %	-	
	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
	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
=1 ≤15	=0	IL 6	CRP
>	=1	≤	Hcy
		=0	ACA
			Logistic
	NIHSS	IL 6	CRP
		Hcy	ACA
			P<
0.05	/	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
	P<0.05
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
	P<0.05
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
	mRS
	NIHSS
	IL 6
	mRS
0.523	0.584
2.5	NIHSS
	ROC
	Hcy
	NIHSS
	IL 6
	AUC

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

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
IL 6 ng/mL	0.759	0.673–0.845	50.72	0.515
CRP mg/L	0.894	0.835–0.953	16.41	0.639
Hcy μg/mL	0.845	0.779–0.910	17.85	0.542
ACA mg/L	0.892	0.839–0.944	0.74	0.617

1.0
0.8
0.6
0.4
0.2

0 0.2 0.4 0.6 0.8 1.0

1 ROC

Figure 1 ROC curve

3

NIHSS

⁹ / IL 6

CRP

–

¹⁰ /

IL 6 CRP

IL 6 CRP

a

IL 6 CRP

P<0.05

NIHSS IL 6 CRP

NIHSS

IL 6 CRP

/

NIHSS

–

¹¹ / IL 6

¹² /

CRP

NIHSS

P<

¹³ /

Hcy
ACA

¹⁴ /

Hcy ACA

¹⁵

Hcy ACA

Hcy ACA

Hcy ACA

Hcy ACA

/

Hcy

¹⁶ / ACA

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

/

IL 6

CRP Hcy ACA

/

NIHSS

–

P<0.05

1

J .

IFN γ TSGF

Sil 2R

1 1 2 1 1

LAEC 2

Sil 2R γ IFN γ TSGF / 2021 3

2023 7 LAEC 106 106

n=53 n=53 /

1 PD L1 1 PD 1 Sil 2R

IFN γ TSGF / ORR DCR

P<0.05 / P>0.05 /

PD L1 PD 1 Sil 2R TSGF IFN γ P<

0.05 /

LAEC

IFN γ Sil 2R TSGF /

Sil 2R IFN γ 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 γ 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 γ 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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				/	P>0.05	/	1 /
				Locally		20210101 /	
advanced esophageal cancer	LAEC						
	LAEC						
	/						
	/						
				¹ /			
				² /			2
	Soluble interleukin 2 receptor	Sil 2R	̄γ				
	Interferon γ	IFN γ					
	Malignant tumor specific growth factor	TSGF					
	-						
	/						
	LAEC				1.2		
	Sil 2R	̄IFN γ	̄TSGF	/		/	260
1					mg/m ² d1 ~d8	75 mg/m ² d2~d4	/
					21 d/1	0	
1.1							
	2021	3	2023	7			
LAEC	106		60.47±6.29	76			
	30		24				
54		28		73			
33			23	56			
27			102	4	/		
	ℓ		2018	№ ³			
	6		KFS	≥70			
	/						
				/			
			106	n=			
53	-	n=53					

2		n %	
Table 2		Comparison of clinical efficacy between the two groups	
n		n %	
		CR	PR
53		1 1.89	21 39.62
53		0 0.00	12 22.64
		SD	PD
53		23 43.40	8 15.09
53		22 41.51	19 35.85
		ORR	DCR
		22 41.51	45 84.91
		12 22.64	34 64.15
		4.289	5.956
P		0.038	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	2 3.77
53		7 13.21	2 3.77	5 9.43	2 3.77	8 15.09	3 5.66	10 18.87	4 7.55
		0.391		0.324		0.759		0.592	
P		0.696		0.746		0.448		0.554	

2.4 Sil 2R IFN γ TSGF IFN γ Sil 2R_h⁻ 2R W
IFN γ TSGF P
Sil 2R TSGF

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IL 1 β sE cad E2

1	1	1	2	3
IL 1β sE cad E2				
/	2020	4	2023	11
BI RADS				
BC	105		BC	BI RADS 4
BI RADS 3	-	PCM	95	PCM /
IL 1β sE cad E2				
IL 1β sE cad E2	BC	PCM	/	BC ROC
Vmax RI PI	PCM	P<0.05 /		IL 1β sE cad E2
-	P<0.05			
P>0.05 /				
AUC 95%CI	-	IL 1β sE cad E2	BC	PCM
E2	P<0.05 /	0.933 0.863 0.966	94.26% 91.77%	IL 1β sE cad
IL 1β sE cad E2				
BC PCM				
/				
IL 1β	E	E2		

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To investigate the significance of serum IL 1 β 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 β 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 β 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 β , 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 β , 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 β , 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 β sE cad E2 Breast ultrasound Plasma cell mastitis Breast cancer

Plasma Cell Mastitis PCM			68			37		
Breast Cancer BC			70			21		
PCM			58.15±10.56 kg			48.32±6.40		
IL 1β			30			P>0.05		
E cadherin sE cad			BC			BC		
Estradiol E2			PCM			-		
IL 1β sE cad E2			1.2			1.2.1		
BI RADS			5			Velocity Maximum		
BC			105			Resistance Index RI		
PCM			95			Pulse Pressure Index PI		
47.45±6.29			40			3 500 r/min		
57.60±10.43 kg			46			10 cm 15 min		
33			28			/		

IL 1 β sE cad

E2 /

1.3

SPSS 23.0 / Shapiro

Wilk /

t /

n % 2 / ROC

IL 1 β sE cad E2 BC PCM

P<0.05 /

2

2.1 IL 1 β sE cad E2

BC IL 1 β sE cad E2

PCM P<0.05 / 1 /

1 IL 1 β sE cad E2

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

	n	IL 1 β pg/mL	sE cad ng/mL	E2 pg/mL
BC	105	10.41 $\pm$ 4.50	8.86 $\pm$ 3.40	205.69 $\pm$ 43.63
PCM	95	1.14 $\pm$ 0.39	5.32 $\pm$ 2.06	95.08 $\pm$ 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 β sE cad E2 BC

PCM

IL 1 β sE cad E2 BC

PCM AUC 0.933

P<0.05 / 4 1 /

3

IL 1 β

7 / IL 1 β

8 /

sE cad

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		
	0 0.00	92 96.84	188.304	<0.001
cm	2.84 $\pm$ 0.85	1.35 $\pm$ 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 $\pm$ 0.53	1.00 $\pm$ 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 $\pm$ 4.64	0.88 $\pm$ 0.45	1.78 $\pm$ 0.69
PCM	95	8.72 $\pm$ 2.40	0.51 $\pm$ 0.33	1.02 $\pm$ 0.41
t		16.904	6.572	9.344
P		<0.001	<0.001	<0.001

4

IL 1 β sE cad E2 BC PCM

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

	AUC	SE	95% CI	%	%
IL 1 β	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 β +sE cad+E2+	0.933	0.045	0.863–0.966	94.26	91.77

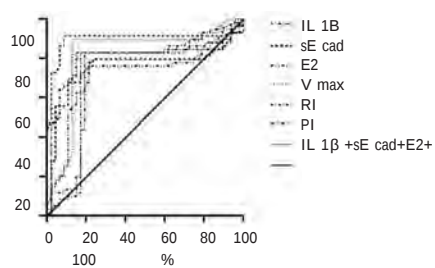
sE cad

10 / E2

11 13 / E2

BC

BC IL 1 β sE cad E2



PCM IL 1β sE cad E2 BC

PCM / IL 1β

BC /

BC
sE cad

/ BC E2 - /

#OU•#\$

SALL4 GS HSP70

4 SALL4⁻ GS⁻ 70
HSP70 / 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

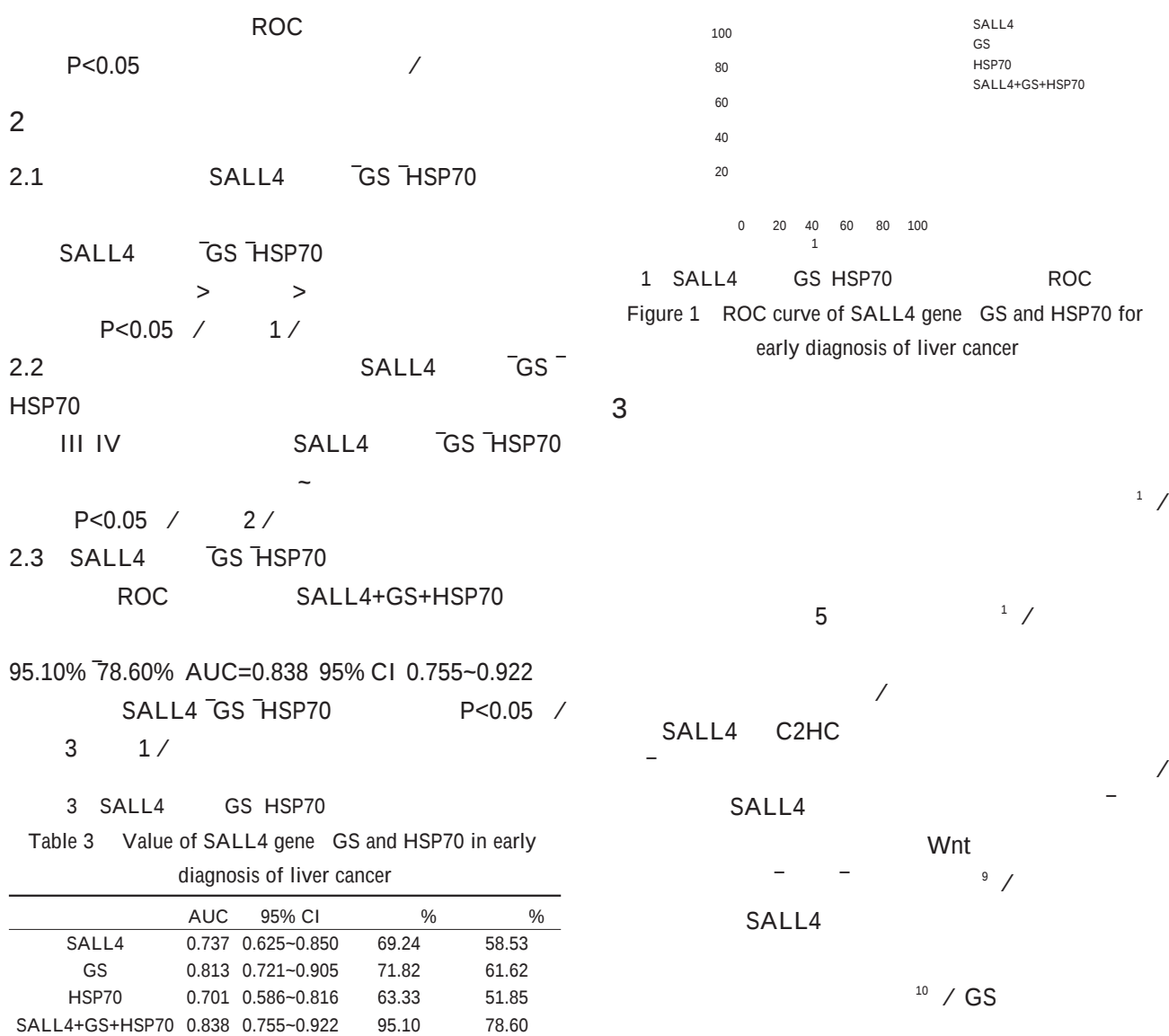


Table 1 Comparison of expression of SALL4 gene GS and HSP70 in different tissues

n	SALL4		GS		HSP70	
	n	%	n	%	n	%
153	11	7.19	142	92.81	0	0.00
141	29	13.47	112	79.43	35	24.82
153	126	82.35	27	17.65	105	68.63
	209.351		209.351		171.539	
P	<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	SALL4		GS		HSP70	
	n	%	n	%	n	%
~	52	31 59.61	21 40.39	28 53.85	24 46.15	21 40.38
~	101	95 94.05	6 5.95	77 76.23	24 23.77	89 88.12
		28.022	28.022	7.993	7.993	38.709
P		<0.001	<0.001	0.004	0.004	<0.001

- 11 / 3 . SCCAg EGFR NSE
GS GS J .
2022 14 9 1511 1514 1518.
12 / GS 4 . GPC3 HSP70 SALL4
AFP J .
2020 42 8 780 782.
5 . D
J .
2010 24 12 1213 1214.
6 .
J . 2002 29 1 83 83.
13 / 7 .
J . 2019
11 9 13 15.
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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1 12 18.
13 .
- - J .
2023 54 6 1208 1218.
14 . N
J . 2020 19 8 898 902.
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mine synthetase and glypican 3 is useful in diagnosis of HBV
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sensitivity J . Eur J Histochem 2018 62 1 2859.
- 12 / GS
HSP70
HSP70
13 / HSP70
HSP70
14 / Moudi 15
HSP70 GS /
ROC SALL4 GS HSP70
SALL4 GS HSP70
1 PDCA
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 β J .

14

Logistic

2021 36 3 433 436.

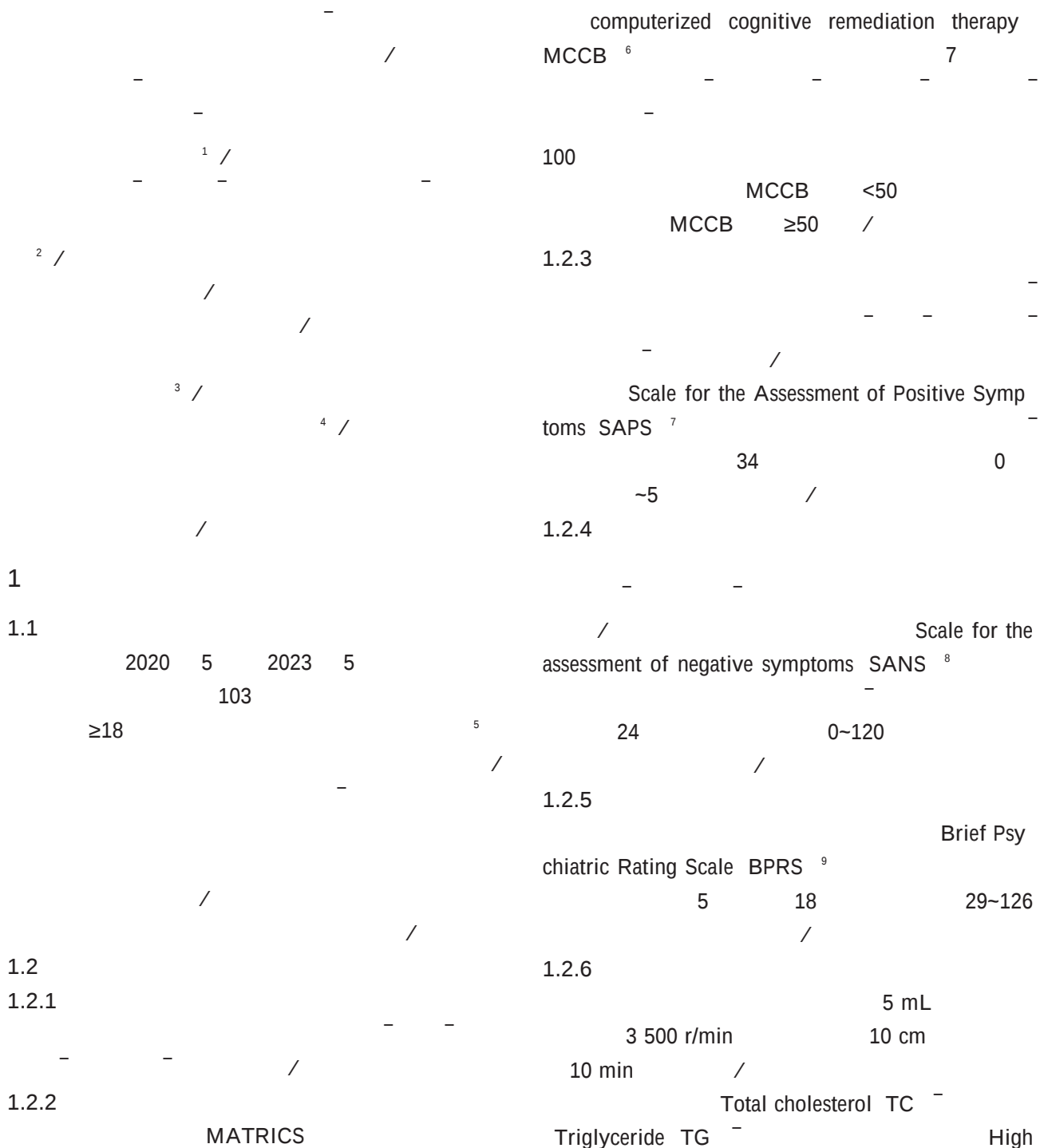
J .

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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



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

1.3

SPSS 22.0

— t n %
2 Logistic

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 /

2.2

SANS
LDL C

P<0.05 / 2 /

2.3

TC TG HDL C LDL C

TC TG HDL C LDL C

1= 0= ROC

3 TC TG HDL C LDL C

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
TG	0.803	0.760–0.916	0.040	2.27 mmol/L
HDL C	0.750	0.763–0.929	0.042	1.44 mmol/L
LDL C	0.833	0.684–0.871	0.048	3.17 mmol/L

1

— n %

Table 1 Comparison of general data between impairment group and non impairment group — n %

	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

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

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 /

3 1 /

ROC

3

¹⁰ /

/ ¹¹

/

/

64.08%

¹²

/

TNFSF15

1		2											
				15 TNFSF15					SNP				
1A TL1A				PBC					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											
ALT AST		t=2.12 2.23 3.35 3.36 P<0.05 TL1A AST ALT											
ALP GGT TBA		r=0.202 0.252 0.313 0.328 0.129 P<0.05 TBIL DBIL ALB											
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		15	



		TNFSF15		SNP		2	
		Haploview4.2		Tagger		2.1	
SNP		/				SNP	
>0.05				r ² ≥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 /	
TL1A				/			
1.2.3				2		TL1A	
						-	
						Table 2 Comparison of TL1A concentrations in peripheral blood between case group and control group	

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

SNP	P		P		P	
rs55717217	AA	54 45.0	85 70.8	16.443	<0.001	A 167 70.4
	AG	59 49.2	31 25.8			
	GG	7 5.8	4 3.3			
rs6478108	CC	20 16.7	35 29.2	7.904	0.019	C 103 42.9
	CT	63 52.5	63 52.5			
	TT	37 30.8	22 18.3			
rs4979462	CC	51 42.5	83 69.2	17.313	<0.001	C 164 68.1
	CT	62 51.7	33 27.5			
	TT	7 5.8	4 3.3			
rs10114470	TT	22 18.3	36 30.0	4.462	0.107	T 110 45.8
	CT	66 55.0	57 47.5			
	CC	22 26.7	27 22.5			
rs1857335	GG	51 42.5	82 68.3	16.233	<0.001	G 163 67.9
	GA	61 50.8	34 28.3			
	AA	8 6.7	4 3.4			
rs12235514	GG	89 74.2	76 63.3	3.445	0.179	G 207 86.3
	GA	29 24.2	40 33.4			
	AA	2 1.6	4 3.3			

10 / TNFSF15

/

45 11 12 /

TL1A TL1A

13 / TNFSF15

9q32

PBC

14 /

rs4979462 - rs55717217 -

PBC

rs4979462 - rs55717217 -

TNFSF15

3' UTR

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 /

TL1A

PBC

PBC	ROC
	P<0.05 /

1/

RNA

15 16

TNFSF15

PBC

TL1A

TL1A

Th1 Th17

3

TL 2 TL 4 TL 12 TL 17 IL 21 13 / 3.5 3.0 2.5
TL1A AST ALT ALP GGT TBA

PBC

ROC TL1A %

9 / IgM PBC

/?

 $\dot{W} \quad R \quad R$

R

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

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 chitecture of primary biliary cholangitis J . Eur J Med
 Genet / F

SAA HMGB1 TNF α

AS A SAA B1
 HMGB1 TNF α AS
 / 2021 1 2023 10 152
 AS Bath BASDAI
 / 80 / C CRP SAA HMGB1 TNF α
 HMGB1 TNF α ESR UA Spearman AS SAA HMGB1 TNF α
 TNF α ROC SAA HMGB1 TNF α
 AS / UA CRP ESR SAA HMGB1 TNF α
 t=5.827 50.112 50.329 35.944 46.683 67.298 P<0.05 /
 SAA HMGB1 TNF α t=10.977 13.302 12.705
 P<0.05 / Spearman AS SAA HMGB1 TNF α
 r=0.743 0.684 0.571 P<0.005 / ROC SAA HMGB1 TNF α
 AS AUC 0.922 92.76% 90.00% P<0.05 / AS
 SAA HMGB1 TNF α SAA HMGB1 TNF α 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 α (TNF α) 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 α , erythrocyte sedimentation rate (ESR) and uric acid (UA) were compared between the two groups. The correlation between serum levels of SAA, HMGB1, TNF α and disease activity was analyzed using the Spearman method. The value of single and combined detection of serum SAA, HMGB1 and TNF α 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 α 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 α 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 α 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 α 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 α in the serum of patients with AS activity were significantly increased. SAA HMGB1 and TNF α 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 α Disease activity

ankylosing spondylitis AS 1 1984 AS
6
2 18 3 6

AS
/ AS
/ AS
AS
 α Tumor Necrosis Factor α TNF α

serum amyloid A SAA

SAA SAA

3 / B1 High Mobility
Group Protein B1 HMGB1

HMGB1
4 5 HMGB1 AS
/ SAA HMGB1
TNF α AS
AS /

1

1.1

2021 1 2023 10
152 AS
80 /

DAI		⁷	BASDAI		
-	-	-	-	-	-

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

SAO

2023 3

VEGF miR 34a HIF 1α

102 SAO

n=51 n=51

VEGF miR 34a HIF 1α

Qm Vm

3.308 3.608 P<0.05

SSS

GFAP

t=3.891 4.654 6.339 P<

0.05

HIF 1α miR 34a

t=6.436 5.591 4.217 P<0.05

²=0.270 P>0.05 /

SAO

HIF 1α miR 34a

VEGF /

miR 34a

1α

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To investigate the efficacy of butaphthalein combined with eEdaravone dextrocarnophorol 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 1α HIF 1α 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 dexcaranol using a random number table method. Cerebral hemodynamics neurological function serum miR 34a VEGF HIF 1α 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 α and miR 34a in the peripheral blood of the combination group was statistically lower than before treatment and in the butylphthalein group and the expression of VEGF was statistically higher than in the butylphthalein 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 buphthalein and Edaravone dextrocamphorol in the treatment of SAO stroke can improve cerebral hemodynamics and cerebral perfusion enhance neurological function reduce HIF 1 α and miR 34a expression and elevate VEGF levels.

Butylphthalein Edaravone dextrocamphenol SAO miR 34a EGF HIF 1 α

Small artery occlusion SAO

2018Ne⁶

25%

SAO

1 / SAO

2 SAO

23 /

JSLC20230003 /

1.2

4 /

4E1

H20100041 100 mL

25 mg 0.9 g 100 mL/

5 /

SAO

Vascular endothelial growth fac

tor VEGF miR 34a

1 α Hypoxia in

ducible factor 1 α HIF 1 α /

2 / /

3~14 d /

1.3

1.3.1

CT

Relative cerebral blood flow rCBF

Mean blood flow

Mean blood flow Qm /

5 mL

3 500 r/min

12.5 cm

10 min

Glial fibrillary acidic protein GFAP /

2022 3 2024 3

102 SAO

51

32 19

0~48 h

5 32 /

34 17

0~48 h

3 31 /

$P>0.05$ /

65.22 $\pm$ 9.94

61.37 $\pm$ 10.80

1.3.3

2.2

SSS

GFAP

Scandinavian

SSS

GFAP

Stroke Scale SSS⁷

SSS

P>0.05

58

/

SSS

GFAP

miR 34a VEGF HIF 1α

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⁻

	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 /

miR 34a

/

95

3 min 95 5s

60

15s

72

15s

40

/

U6

miR 34a

5' TCGCGCTG

GCAGTGTCTTAGCT 3'

5' CCAGTG

CAGGGTCCGAGGTATT 3' U6

5' CTC

GCTTCGGCAGCACA 3'

5' AAC

GCTTCACGAATTTGCGT 3'

2^{ΔΔCt}

2.3

VEGF miR 34a HIF 1α

miR 34a

/

VEGF miR 34a⁻

ABclonal

VEGF⁻

HIF 1α

P>0.05

HIF 1α

/

HIF 1α miR 34a

1.3.4

VEGF

P<0.05 / 3 /

2.4

1.4

SPSS 26.0

/

-

P>0.05 / 4 /

t

3

t

n %

SAO

2

/

P<0.05

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2

2.1

rCBF Qm Vm

-

-

8 /

rCBF Qm Vm

P>0.05

-

-

P<0.05 / 1 /

9 /

1

rCBF Qm Vm

-

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

	n	VEGF pg/mL		miR 34a		HIF 1α 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
	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
t		0.464	5.591	0.439	6.436	0.054	4.217
P		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	2 3.92	2 3.92	1 1.96	8 15.69
	51	1 1.96	2 3.92	1 1.96	1 1.96	2 3.92	2 3.92	1 1.96	10 20.00
									0.270
P									0.604



VEGF

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- 12

FIB RBP NAG TRF

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

Henochschonlein purpura nephri
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 Urinary
 retinol binding protein RBP
 N β _FS A $\mathbb{L}$ A u $\mathbb{L}$ qB $\pm$ = F TetctA et n qB $\pm$ 4 Π r X s

P<0.05			/		2.4	FIB	RBP	NAG	TRF
2					ROC			FIB	RBP
2.1					NAG		TRF		
	-	-		-	AUC	0.930		0.833	
HSP			P>0.05		0.860		P<0.01	/	3 /
	FIB	TRBP	NAG	TRF					
			P<0.05	/		1 /			
	n=57		n=114						
	96	30	52.63	66	57.89				
	75	27	47.37	48	42.11				
			7.84±3.32		8.68±3.47				
m ² /kg			15.53±2.48		16.01±2.31				
	52	16	28.07	36	31.58				
HSP	119	41	71.93	78	68.42				
	30	11	19.30	19	16.67				
	141	46	80.70	95	83.33				
FIB g/L			4.78±0.89		1.69				
RBP mg/L			52.63±3.20		68.52±4.4				
NAG U/L			58.10±5.43		63.59±3.40		1.57±0.10	1.1	>
TRF g/L			1.59±0.42		1.1	3			
			HSP			-	-		
2.2			FIB		TRBP	NAG		HSPN	
TRF							/		HSPN
							/		HSPN
	FIB		TRBP	NAG	TRF	9 /			
	<0.05		/		2 /				
			HSPN		100 000		10 /		1.3/
					11 /		HSPN		
HSP									
			/		HSPN				
					HSPN				
								/	
2.3	FIB		TRBP	NAG	TRF	HSPN			
							1 ~7	HSPN	
							12 /		
	Spearman		FIB		TRBP		13 /		FIB
NAG	TRF	HSPN	NAG		TRF	HSPN	FIB		
	r=0.360	0.666	0.400	0.239	P<0.05	/	/ FIB		

[illegible]

AMPK/mTOR / TNF α
/ Elisa
TUNEL
WB AMPK/mTOR AMPK p AMPK mTOR p mTOR
/ 0 ng/mL 1 ng/mL TNF α p AMPK
10 ng/mL TNF α P<0.05 AMPK
P>0.05 / TNF α IL 1 β p AMPK/AMPK
p mTOR/mTOR P<0.05 /
TNF α AMPK/mTOR /
AMPK/mTOR

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Fujian China 352100

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injury by activating AMPK/mTOR pathway.

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

	-	-	-	GAPDH	mTOR	Monoclonal	-
				Phospho mTOR Ser2448	Monoclonal		-
	12	1 /		AMPK Alpha Polyclonal	Phospho AMPKα		ECL
	2 /			Thr172			
					Thermo		-
				-			PBS
	/			-			/
				1.2			
			3 /	1.2.1			
				HNEpC	RPMI 1640		
				+10% FBS+1% PBS		37 °C	5%
	/ AMP	/		CO ₂	70%-80%	0.25%	
AMP activated protein kinase/mammalian target of rapamycin AMPK/mTOR				1.2.2		/	
			4 / AMPK	TNF α		0.1-100 ng/mL	
-	/ mTOR	-	-	TNF α		24 h	
				WB	AMPK p AMPK		TNF α
5 /					/		
AMPK/mTOR			/		/		
1				1.2.3		enzyme linked immuno	
1.1				sorbent assay	ELISA		
	-					20 cm	
				3 000 rpm	15 min		
		-	-		450 nm	OD	/
					OD	TNF α	IL 1β
			-				/
			-	1.2.4		dUTP	
-					Terminal deoxynucleotidyl Trans		
	-		/	ferase Mediated Nick End Labeling	TUNEL		
					4%		20
HNEpC				min 3	H ₂ O ₂		Triton 100
/		RPMI 1640	-	10 min / TUNEL		1 h	
1β ELISA	-		α ELISA	37		5 min /	DAPI
Tunel	BCA						
50T	-					/	
FBS	-	P/S	-			/	
Gibco			DAPI	1.2.5	Western Blot	WB	
							20 cm

4 3 000 rpm 15 min 2.2

BCA

loading buffer 98 5 min

SDS PAGE 4 2 /

GAPDH 1Æ10 000 AMPK 1Æ2 000 p AMPK 1Æ

1 000 mTOR 1Æ5 000 p mTOR 1Æ2 000

TBST 3 2 h TBST 3 /

1.3

Prism 8.0 GraphPad USA

t / P<0.05 /

2

2.1 TNF α AMPK

TNF α HNEpC 0 ng/mL

1 ng/mL 10 ng/mL 100 ng/mL AMPK

P>0.05 10 ng/mL

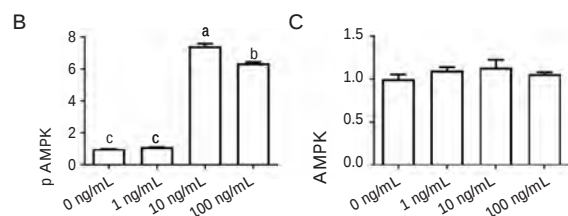
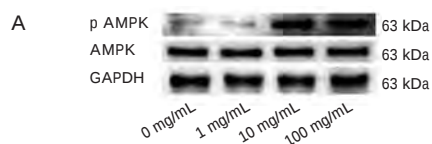
p AMPK 0 ng/mL 1 ng/mL

100 ng/mL p AMPK 0 ng/mL

100 ng/mL 10 ng/mL

P<0.05 / 0 ng/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 /

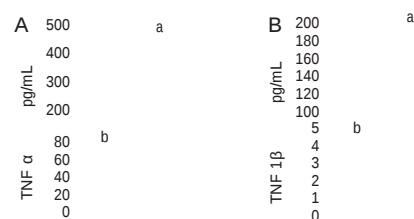
1 TNF α AMPK

p AMPK

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

TNF α IL 1β

P<0.05



A TNF α B IL 1β

P<0.05 / a P<0.05 b

2 ELisa TNF α IL 1β

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

2.3

P< 0.05 / 3 /

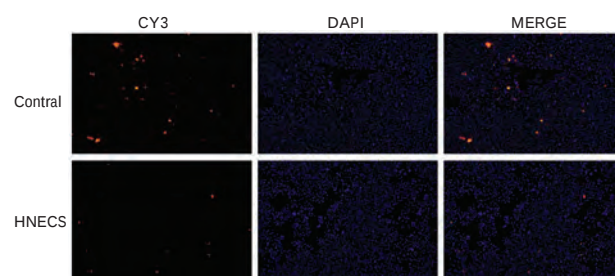


Figure 3 TUNEL detection of apoptosis fluorescent TUNEL staining ×100

2.4 AMPK/mTOR

AMPK mTOR

P>0.05

p AMPK/AMPK

P<0.05

p mTOR/mTOR

P<0.05 / 4 /

3

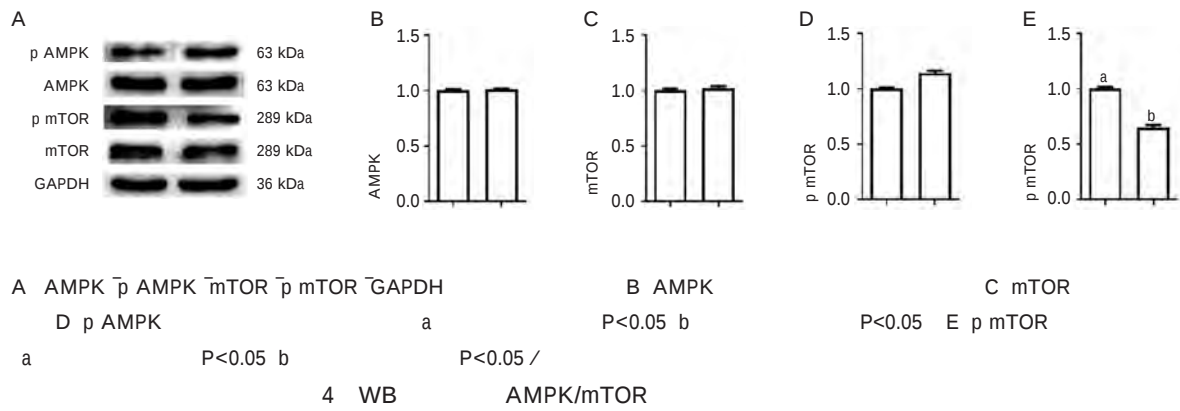


Figure 4 Expression of AMPK/mTOR signaling pathway related proteins detected by WB

/

AMPK

AMPK

AMPK

AMPK

10ng/mL TNF α

AMPK / TNF α

TNF α IL 1 β

SIRS

IBD

AMPK

AMPK

/ p AMPK

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F ¶

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Multivariate logistic regression analysis showed that hypoproteinemia external ventricular drainage duration >7 days and increased expression levels of NF κ B PCT and IL 1 β 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 κ B PCT IL 1 β 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 κ B PCT and IL 1 β 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.

External ventricular drainage Intracranial infection PCT NF κ B IL 1 β

External Ventricular Drainage

EVD

- -

¹ / EVD

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EVD

² /

EVD

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Procalcitonin PCT

EVD

³ /

Interleukin

IL 1 β

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IL 1 β

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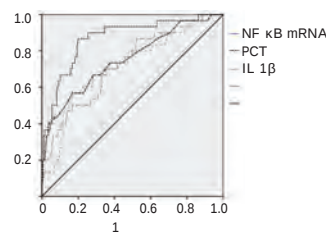


Figure 1 ROC curve analysis of intracranial infection after EVD

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TLR4 NF κB

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

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To investigate the effect of Huangqi Yishen granule combined with sevelamer on the efficacy and Toll like receptor 4 TLR4 and nuclear transcription factor κB NF κB 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 α TNF α TLR4 NF κB 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 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 α 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 κ B mRNA levels between the two groups before treatment $t=1.526$ 0.622 $P>0.05$. After treatment TLR4 and NF κ 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 Yishen 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 κ B expression.

. MHD Huangqi Yishen Granules Micro inflammatory state TLR4 NF κ B

Maintenance hemodialysis MHD				1			
				-			
				/			
				n=66 /			
				28 24			
				n=52			
				<6			
				60.14±5.38			
				29			
				20			
				3			
				22.48±8.69			
				/ 5 1 3			
				61.75±7.60			
				33			
				MHD			
				26 7			
				23.57±7.08			
				/			
				MHD			
				6			
				/			
				7			
				MHD			
				/ Toll			
				4 Toll like			
				receptor 4 TLR4 TLR			
				8 /			
				κB nuclear factor kappaB NF κB			
				MHD			
				NF κB			
				9 /			
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				1			
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				2019 1 2022 11			
				118 MHD			
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				>6			
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				/			
				-			

1.3.2

2.4 TLR4 NF κ B /
TLR4 NF κ B mRNA
P>0.05 TLR4 NF κ B

mRNA

P<0.05 / 4 /

4 TLR4 NF κ B

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

	n	TLR4 mRNA		NF κ 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

6.06%

4/66

3.85% 2/52

P>

0.05 / 5 /

5 n %

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 /

n N

MHD

/

- -

- -

-

/ -

-

/

/ -

MHD

¹⁴

- -

-

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-

/

¹⁵

MHD

MHD

MHD

IL 6 TNF α

¹⁶ NLR

MHD

^{17 18} /

/

TLR4 NF κ B

/

/

MHD

TLR4 NF κ B

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19

21 1 ALI PLR

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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
		^a P<0.05	^b P<0.05	^c P<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

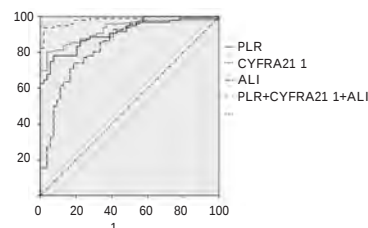
2 n %

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

0.982

P<0.05 / 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

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
				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

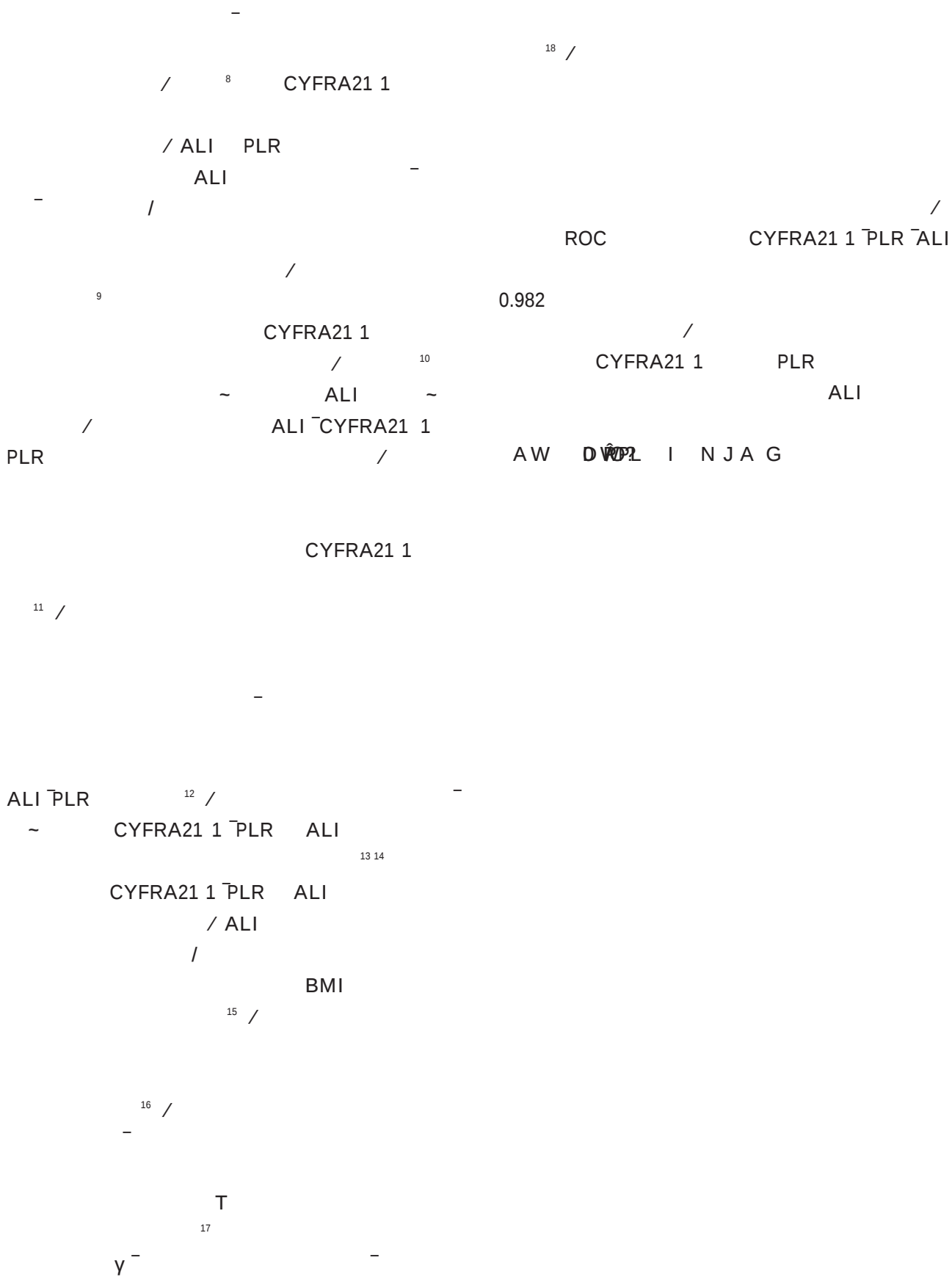
4 PLR ALI CYFRA21 1

ROC

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

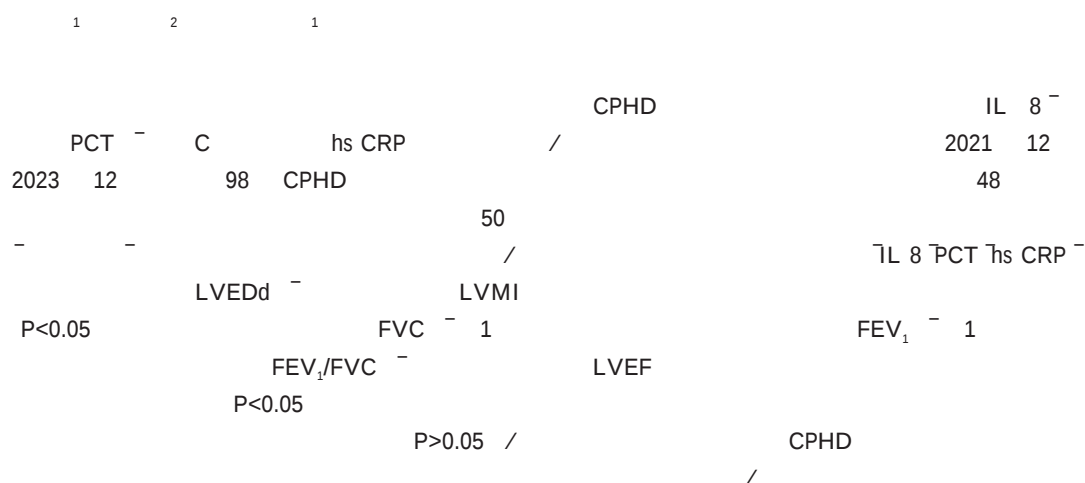
			%	%	95%CI	AUC	P
PLR	163.36	0.707	78.1	92.6	0.858–0.951	0.904	<0.001
ALI	44.01	0.555	74.0	81.5	0.779–0.913	0.846	<0.001
CYFRA21 1	7.49 ng/mL	0.765	80.2	96.3	0.884–0.967	0.926	<0.001
LDH+SAA+IL 17A		0.919	93.8	98.1	0.966–0.999	0.982	<0.001

3



hs CRP

IL 8 PCT

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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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Figure 1: A forest plot showing the association between CPHD and various outcomes. The plot displays odds ratios (OR) and 95% confidence intervals (CI) for different studies. The outcomes are: Chronic pulmonary heart disease (CPHD), New York Heart Association (NYHA), and Mortality. The plot includes a vertical line at OR=1.0, indicating no association. The plot is divided into three sections: CPHD, NYHA, and Mortality. The plot shows that CPD is associated with a higher risk of CPHD, NYHA, and Mortality. The plot also shows that CPD is associated with a higher risk of CPHD, NYHA, and Mortality. The plot includes a vertical line at OR=1.0, indicating no association. The plot is divided into three sections: CPHD, NYHA, and Mortality. The plot shows that CPD is associated with a higher risk of CPHD, NYHA, and Mortality. The plot also shows that CPD is associated with a higher risk of CPHD, NYHA, and Mortality.

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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 higher than that of the control group.

2.3	MMP2	TMP1	VEGF	P>0.05
	MMP2	TMP1	NPWT	P<0.05 / 4 /
		TMP1	MMP2	P<0.05 /
	P<0.05 /	VEGF	1 /	
	3	n %		

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

4		MMP2	TMP1	VEGF	-	
Table 4	Simple effect analysis of MMP2 TMP1 and VEGF levels at different time points between the two groups					
	n	0 d	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		

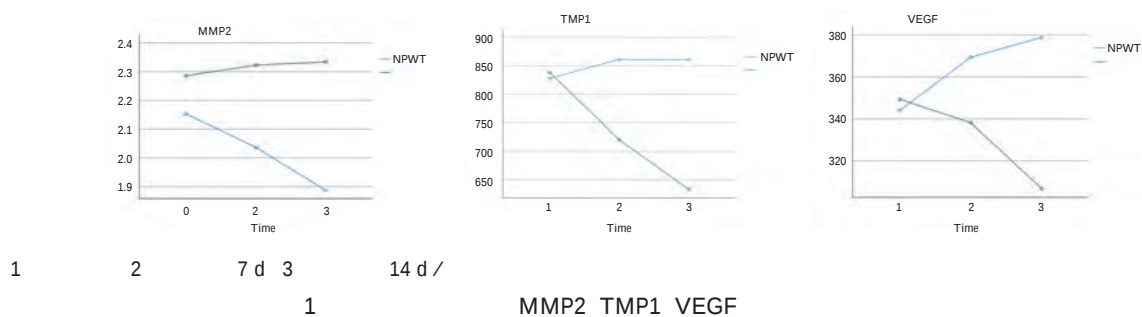


Figure 1 The interaction and levels of MMP2 TMP1 and VEGF at different time points between the two groups

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

Table 6	Comparison of follow up between the two groups					
	n	3	6	n	%	
	15	0 0.00 ^a	0 0.00	3	20.00	0 0.00 ^a 1 6.67
	15	3 20.00	2 20.00	1 6.67	4 26.67	3 20.00 0 0.00
		4.493	4.493	<0.001	0.536	4.493 <0.001
P		0.034	0.034	1.000	0.464	0.034 1.000

^aP<0.05 /

- | Study | Intervention | Control | Outcome | Significance |
|-------|---|--|----------------------------------|-------------------------|
| 11 | NPWT | DFO | 14 d CRP | |
| 12 | NPWT | DFO | 7 d 14 d MMP2 | |
| 13 | TIMP 1 | MMPs | 6 / VEGF | |
| 14 | NPWT | DFO | VEGF | |
| 15 | NPWT | DFO | VEGF | |
| 1 | J . | 2019 | 21 7 636 640. | |
| 2 | J . | 2022 | 36 6 604 607. | |
| 3 | Garrett M Gray S et al. | Audit of diabetes related lower extremity amputations in the Northern Region of New Zealand | 2013 2016. J . New Zealand Med J | 2024 1598 7 44 54 |
| 4 | J . | 2019 | 20 4 207 229 | |
| 5 | J . | 2022 | 30 2 189 192. | |
| 6 | J . | 2021 | 50 8 155 | |
| 7 | J . | 2021 | 26 | |
| 8 | J . | 2024 | 44 7 39 41. | |
| 9 | J . | 2020 | 22 10 787 790 | |
| 10 | J . | 2021 | 35 9 900 905. | |
| 11 | Yong E Gong H Liew H et al. | Getting a Foothold on Diabetic Foot Disease Outcomes of a Multidisciplinary Clinical Pathway for Inpatient Diabetic Foot Care A 17 Year Institutional Review | J . Int J Low Extrem Wounds | 2023 15347346231183740. |
| 12 | Chang L Duan W Chen A et al. | Preparation of polyacrylonitrile based fibres with chelated Ag ions for antibacterial applications. | J . R Soc Open Sci | 2020 7 7 310 324. |
| 13 | J . | 2021 | 41 2 5 7. | |
| 14 | Tallapaneni V Kalaivani C Pamu D et al. | Acellular Scaffolds as Innovative Biomaterial Platforms for the Management of Diabetic Wounds | J . Tissue Eng Regen Med | 2021 18 5 713 734. |
| 15 | Zhang WQ Tang W Hu SQ et al. | Effect of matrix metalloproteinases 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 $\chi^2/t=11.953$ 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

Multiple Myeloma MM
/ - AML 2017 020 /
1.2
1.2.1
1 / MM / 2
MM
/ Th17 / - -
21 Interleukin 21 IL 21 M IgG IgG -
MM international staging system ISS -
2 / Lactate Dehydrogenase IL 21 -
LDH NAD LDH 1q21 /
1.2.2
3 / 1q21 FISH
MM /
4 / 3 mL 2 mL EDTA
1q21 - IL 21 LDH IL 21 Q β EP
MM LDH MM /
MM
1
1.1
2017 10 2023 6
MM 162
/
2022 No⁵ 33~92
/
/

113 - 49 / 3

1 -

1.3

SPSS 22.0

t n %

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

LDH

0.05 / 2 /

2

n %

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

	n=162	n=64	n=98	/t	P
				7.150	0.008
>60	99	31 48.44	68 69.39		
≤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

/

-

P<

0.05 / 2 /

3			MM						
Table 3 Multivariate regression analysis of independent risk factors influencing the prognosis of MM patients									
				S.E	Wald	OR	95% CI	P	
ISS	~	=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
		=0	=1	0.742	0.483	2.360	2.100	0.815~5.412	0.124
		=0	=1	0.531	0.201	6.979	1.701	1.147~2.522	0.008
1q21				0.683	0.218	9.816	1.980	1.291~3.035	0.002
IL 21 pg/mL				0.730	0.228	10.251	2.076	1.327~3.244	0.001
LDH U/L									

IL 21
*

• •

IncRNA PVT1 HIF 1 α

Q19039

1. 610011
2. 518000

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group the expression levels of serum lncRNA PVT1 and HIF 1 α in EMs patients were obviously higher with statistical significance $P<0.05$. As the progression of R AFS stage the expression levels of serum lncRNA PVT1 and HIF 1 α in EMs patients gradually increased $P<0.05$. Pearson correlation analysis showed that lncRNA PVT1 was positively correlated with HIF 1 α $r=0.419$ $P<0.05$. Spearman correlation analysis showed that the levels of serum lncRNA PVT1 and HIF 1 α were obviously positively correlated with the R AFS stage of EMs patients $r=0.467$ 0.699 $P<0.05$. The ROC curve showed that the AUC of lncRNA PVT1 and HIF 1 α in diagnosing EMs alone was 0.915 and 0.873 the sensitivity was 87.0% and 73.9% and the specificity was 70.7% and 73.9% respectively the AUC of the combined diagnosis of EMs was 0.962 the sensitivity was 87.0% and the specificity was 78.3%. The proportion of patients with high expression of lncRNA PVT1 and HIF 1 α in clinical stage to deep growth cyst size ≥ 3 cm and dysmenorrhea was significantly higher than those with low expression of lncRNA PVT1 and HIF 1 α and the difference was statistically significant $P<0.05$.

The expression levels of serum lncRNA PVT1 and HIF 1 α in patients with EMs are increased and the high expression of lncRNA PVT1 and HIF 1 α is related to the R AFS stage. Therefore the combined detection of the two may be beneficial for the early targeted treatment in clinical settings.

Endometriosis lncRNA PVT1 HIF 1 α

endometriosis EMs 7 3

EMs /

10%~15% -

12 / /

EMs 92

-

3 / EMs 35.73 $\pm$ 9.47 /

EMs EMs /

/

1.2

1.2.1 lncRNA PVT1 HIF 1 α

lncRNA PVT1

EMT

-

4 / PCR qRT PCR / 3~5 mL

LncRNA - 12 cm 3 000

ceRNA - rpm 20 min -80 /

EMs /

1 PVT1 LncRNA

Invitrogen RNA TriZol

cDNA qRT PCR 1 /

2

5 /

EMs lncRNA PVT1 HIF 1 α /

1

1.1

2021 10 2023 7

92 EMs EMs

34.86 $\pm$ 9.32

R AFS 6 EMs

16 25 38 13 /

1

No

1 PCR

Table 1 PCR primer sequence

	5' 3'	5' 3'
IncRNA PVT1	TTGGCACATACAGCCATCAT	GCAGTAAAAGGGGAACACCA
GAPDH	GAAGGTGAAGGTCGGAGTC	GAAGATGGTGATGGGATTTC

	n %	²	4	R AFS	IncRNA PVT1	HIF 1α
Spearman	EMs	IncRNA				
PVT1 HIF 1α	R AFS					
ROC	IncRNA PVT1	HIF 1α				
EMs	Z	IncRNA				
PVT1 HIF 1α		EMs				
ROC	/ P<0.05	/				

2

2.1

EMs

P<0.05

BMI

P>0.05 / 2 /

2.2

HIF 1α

0.05 / 3 /

3

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

	n	IncRNA PVT1	HIF 1α 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

2.3

PVT1 HIF 1α

R AFS

IncRNA PVT1 HIF 1α

P<0.05

R AFS

EMs

IncRNA PVT1 HIF 1α

P<0.05 / 4 /

2

n %

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	35 38.04
/t		0.628	0.459	1.094
P		0.531	0.647	0.269

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

	n	IncRNA PVT1	HIF 1α 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

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

2.4

IncRNA PVT1

HIF 1α

R AFS

Pearson

HIF 1α

r=0.419 P<0.05

Spearman

PVT1 HIF 1α

R AFS

r₁=0.467 r₂=0.699 P<0.05 /

2.5

IncRNA PVT1 HIF 1α

EMs

ROC

IncRNA PVT1 HIF 1α

EMs

AUC

0.962 95%CI 0.940~

0.985

87.0%

78.3%

AUC

IncRNA PVT1

AUC Z=2.015 P=0.044

HIF 1α

AUC Z=2.836 P=0.005 /

1 /

2.6

IncRNA PVT1 HIF 1α

EMs

IncRNA PVT1=1.35

HIF 1α=66.99 pg/mL

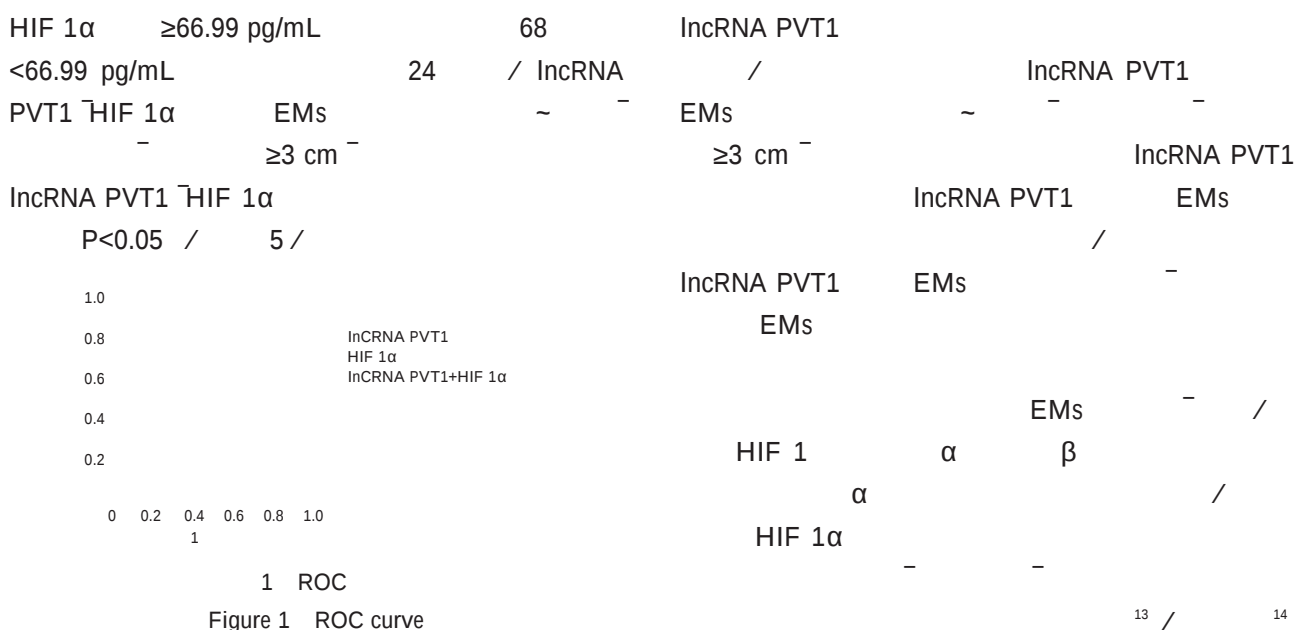
IncRNA PVT1

≥1.35

81

<1.35

11



3

EMs HIF 1α /

8 / EMs

/ EMs

9 /

EMs

10 /

EMs

ROC

IncRNA

EMs

PVT1 HIF 1α

IncRNA PVT1 HIF 1α EMs

IncRNA PVT1 HIF 1α

11 / Kong 12

IncRNA PVT1

EMs

IncRNA PVT1

		IncRNA PVT1		HIF 1α		EMs		n %			
		n		P		n		P			
		n=81		n=11		n=68		n=24			
~	41	32	39.5	9	81.82	5.410	0.020	36	52.94	5	20.83
~IV	51	49	60.49	2	18.18			32	47.06	19	79.17
	50	40	49.38	10	90.91	6.731	0.009	31	45.59	19	79.17
	42	41	50.62	1	9.09			37	54.41	5	20.83
<3 cm	40	31	38.27	9	81.82	5.806	0.016	24	35.29	16	66.67
≥3 cm	52	50	61.73	2	18.18			44	64.71	8	33.33
	54	52	64.20	2	9.09	6.667	0.010	34	50.00	20	83.33
	38	29	35.80	9	90.91			34	50.00	4	16.67

PDK1

1 2 2

CHF 3 1 PDK1

2022 3 2024 3 180 CHF

CHF NYHA

110 / PDK1 mRNA C

CRP - suPAR - 6 IL 6 - 8 IL 8 /

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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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 and severe heart failure with grade to . 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

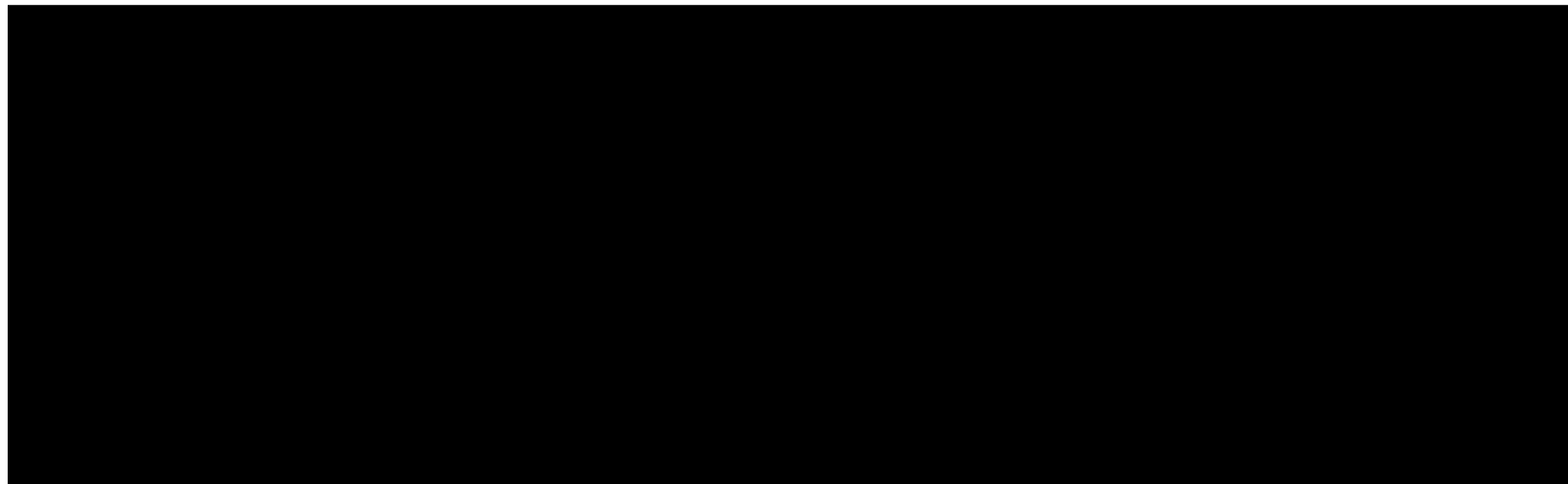
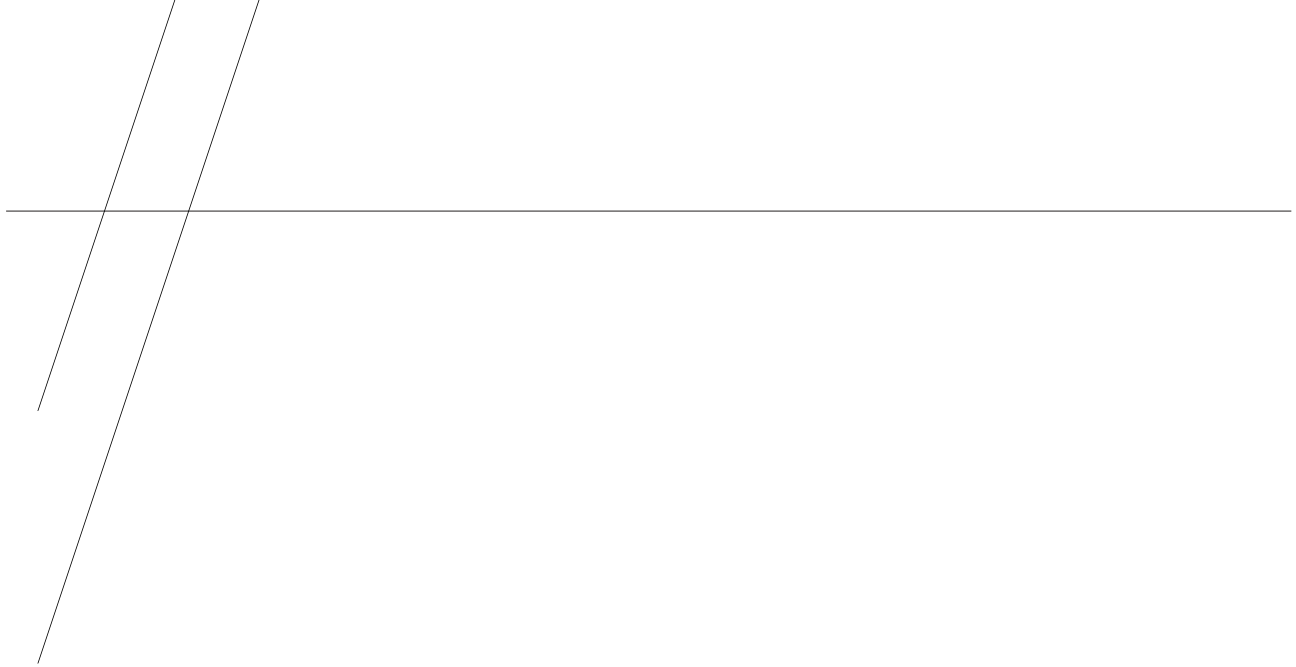
2. 610000

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CRP suPAR IL 6 and IL 8 in serum were higher than those in the control group with statistical significance $t=12.193$ 19.347 18.742 13.044 $P<0.05$. The mRNA expression level of PDK1 in the peripheral blood of patients with severe heart failure in the CHF group was lower than that of patients with mild heart failure $t=9.925$ $P<0.05$ and the serum levels of CRP suPAR IL 6 and IL 8 were higher than those of patients with mild heart failure with statistical significance $t=5.444$ 5.647 6.312 8.015 $P<0.05$. The mRNA expression level of PDK1 in the peripheral blood of the CHF group was negatively correlated with the levels of CRP suPAR IL 6 and IL 8 in serum $P<0.05$. The expression of PDK1 in the peripheral blood of CHF patients is decreased which is related to the worsening of heart failure and the activation of the inflammatory response.

Chronic heart failure 3 phosphoinositide dependent protein kinase 1 Inflammatory response

chronic heart failure CHF	56.97±4.95	22.97±2.76 kg/m ² /
-	0.05 /	/
-	1.2	
1.2 /	1.2.1	PDK1 mRNA
CHF		
/ 3	1 3 phosphoinosit	5 mL Trizol RNA
ide dependent protein kinase 1 PDK1	1 µg RNA	.
	PDK1	
	PDK1	
-	/ CHF	
-	PDK1	
PDK1	3 / CHF	
PDK1	/	
1		
1.1		
2022 3 2024 3		
180 CHF		
	CHF	
4	New York Heart Associa	
tion NYHA	~ 5	
	/	
-	-	-
	/ 180 CHF	
CHF	104 - 76	
58.36±4.96	23.18±2.85 kg/m ² /	
	110	
58	- 52	



PCR PCR
PCR - PCR
8 /
/
- /
1 / - - - / -
PCR - - - / /
mNGS 23 / - / /
DNA /
4 / 2.2.2 /
PCR - -
PCR / -
5 / -
2 mNGS
2.1 /
/
- - /
/
2.2 / /
2.2.1 / mNGS -
- - - /
- - - - / - -
6 / - /
- - / -

$\bar{\text{DNA}}$

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ER β
15 /
HSD17B6
17 β HS
Ds HSD17B6
HSD17B6
/
3 HSD17B6
3.1 HSD17B6
ovarian syndrome PCOS
PCOS
- 5 5 N Q

	HSD17B6	27 /
1	Programmed death re	
ceptor 1 PD 1		
	PD 1	HSD17B6 HSD3B1 CYP19A1
β 1 3 N	3	
23 /		28 /
HSD17B6	LUAD	
LUAD		/
	5	
/		HSD17B6
4.3 HSD17B6 PCa		
HSD17B6 PCa	DHT	
/	HSD17B6	
DHT	HSD17B	-
6 HSD17B10		
DHT		HSD17B6
PCa	7 24 /	
PCa	PCa	
/		HSD17B6
PCa	HSD17B6	
3 α /3 β diol	DHT PCa	/
HSD17B6		
2	HSD17B6	-
HSD17B6		/
	HSD17B6	
PCa	25 /	
HSD17B6	PCa	-
3 α / β	DHT	
DHT		/
4.4 HSD17B6		
HSD17B6		
	Non small	
cell lung cancer NSCLC		
/ NSCLC		
HSD17B6 NSCLC		
26		
HSD17B6	/ HSD17B6	

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2009 5

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