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Volume 12 Number 4 April 2020

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Figure P431 The result of immortalized lymphocyte cell line transformation on day 0 day15 and day 30 x50

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分子诊断与治疗杂志

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RNA GAS5

1 2 3 1 1*

RNA LncRNA GAS5 LncRNA 200 nt

2 5 4 087 nt 12 LncRNA GAS5

LncRNA GAS5

The Role Of LongNon - Coding RNA GAS5 In The Occurrence And Development Of Atherosclerosis And Atherosclerosis-related Diseases

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ABSTRACT LongNon Coding RNA LncRNA is a type of RNA that is located in the nucleus or cytoplasm and is longer than 200 nt. LncRNA GAS5 is a member of the LncRNA family that has attracted attention in recent years. The encoding gene is in the q25 regio of human chromosome 1, has a total length of 4 087 nt and consists of 12 exons. Several studies have shown that LncRNA GAS5 is involved in inflammation mediates vascular endothelial cell damage and plays an important role in the development of atherosclerosis and related diseases. This article reviews the role of LncRNA GAS5 in the occurrence and development of atherosclerosis and atherosclerosis related diseases.

KEY WORDS LncRNA GAS5 Atherosclerosis Coronary heart disease Diabetes

RNA long non coding RNA

LncRNA

200 nt

RNA ¹

LncRNAs

2018GXNSFAA138120

81560552

2018AB58018

1. 541000
2. 530000
3. 533000

* E-mail wyshi22@163.com

h qRT PCR 96
LncRNA GAS5
LncRNA GAS5 2
LncRNA GAS5 12

qRT PCR 0.81 LncRNA GAS5
2 85.1% 67.3%
2

LncRNA GAS5
³⁴

LncRNA GAS5 2
Shi ³⁵ 2
LncRNA GAS5 LncRNA GAS5

LncRNA GAS5
B
LncRNA GAS5
2
5

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was statistically significant $P < 0.05$. There are 13 male suspects and 7 females in this study. respectively no statistically significant difference in gender positive detection rates $P > 0.05$. The results from different sample types showed that the positive detection rate of throat swabs was the lowest nasopharyngeal swabs and sputum samples were higher than throat swabs. Interestingly the positive detection rate of fecal samples was the highest although the sample number in this group was small compared to other types. However there was no significant difference in the positive rate of the same suspected patient with different sample types at the same time. Conclusion The RT-PCR method has different positive detection rates for different sample types which can provide guidance for the selection of samples in laboratory testing and better prevention and control of epidemics.

KEY WORDS Real Time Polymerase Chain Reaction Positive rate SARS-CoV-2 throat swabs

Severe Acute Respiratory Syn
drome Coronavirus 2 SARS-CoV-2

1
2020 3 26 SARS-CoV-2
8
37
2 SARS-CoV-2
SARS-CoV-2 3

0.05 132 P> P>0.05 3

Table 3 SARS CoV 2 nucleic acid detection results at the same time in the same suspected patient with different sample types

		n %		χ^2	P
303	303	0 0.00	303 100.00	1.820	1.000
	303	0 0.00	303 100.00		
	303	1 0.33	302 99.67		
	909	1 0.11	908 99.89		
4	4	0 0.00	4 100.00	/	/
	4	0 0.00	4 100.00		
	4	0 0.00	4 100.00		
	12	0 0.01	12 100.00		
280	280	7 2.50	273 97.50	0.079	0.788
	280	6 2.14	274 97.86		
	560	13 2.32	547 97.68		
132	132	10 7.58	122 92.42	0.733	0.521
	132	14 10.61	118 89.39		
	264	24 9.09	240 90.91		

3

CoV 2 SARS RT PCR 50%¹³ SARS CoV 2 30% ~

SARS CoV 2 SARS CoV 2 SARS CoV 2

9 / SARS CoV 2

14 SARS CoV 2

SARS CoV 2 SARS

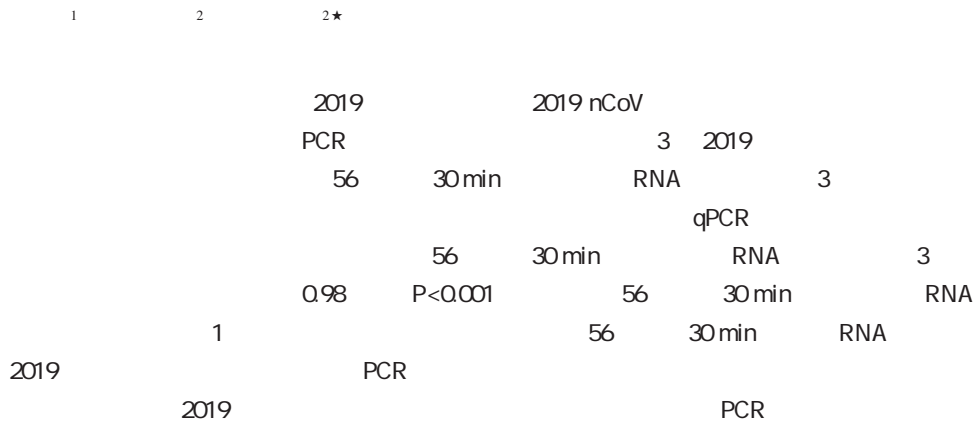
Liu⁹ Rui CoV 2¹⁵

10 / 56.25%¹⁶

4 1

• COVID 19

2019



The effect of 2 different inactivation methods on the results of viral nucleic acid in 2019 new coronavirus throat swab specimens

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1. Laboratory of Puyang Oilfield General Hospital Puyang Henan China 457001 2. Laboratory of Puyang people sHospital Puyang Henan China 457000

ABSTRACT Objective To investigate the effect of two different pharyngeal swab specimens inactivation methods on the results of 2019 new coronary virus fluorescent quantitative PCR. Method Three cases of 2019 new coronary virus positive specimens were treated by two different inactivation methods the nucleic acids were extracted by manual centrifugation method from three groups of samples which were inactivated at 56 for 30 minutes and inactivated with guanidine salt RNA preservation solution. Simultaneous detection of virus content with two different nucleic acid detection kits the results of qPCR were analyzed statistically. Result The results of two different nucleic acid detection reagents showed that the results of the three groups which were not inactivated inactivated at 56 for 30 minutes and inactivated with guanidine salt RNA preservation solution were significantly correlated and had good consistency. The correlation coefficients among the three groups were all above 0.98 P<0.001 . Conclusion 2019 new coronary virus nucleic acid qPCR results were not affected by the two inactivation methods

KEY WORDS 2019 new coronary virus Novel coronary virus pneumonia qPCR

2019 2019 novel coronavirus 10 2019 nCoV
 2019 nCoV RNA WHO
 29 903 bp 10 Novel coronavirus pneu

1. 457001
 2. 457000

* E-mail mengqfoo@163.com

monia NCP

RNA

1.2

1.5

PCR

PCR

3

1.6

Graphpad Prism 8.0

Spearman

Bland Altman

CT

CT

PCR

2

1

2.1 3 qPCR

A

56

30 min

1.1

CT

Spearman

r=

2020 2 1

0.9959 P<0.001

RNA

3

CT

r=0.9958 P<

0.001 56 30 min

RNA

CT

r=0.9966 P<

1.2

2019 nCoV

0.001 1 B

56

30 min CT

Spearman

RNA

r=0.9886 P<0.001

RNA

RT PCR

CT

r=

A

0.9967 P<0.001 56 30 min

RNA

B RNA

CT

r=

0.9915 P<0.001 2

A

B

PCR ABI7500 Life Technologies NY

min R r

56

30

USA

1.3

3

3

2-3 mL

140 L μ

280 L μ

56 30 min

280 L μ

280 L μ RNA

1.4

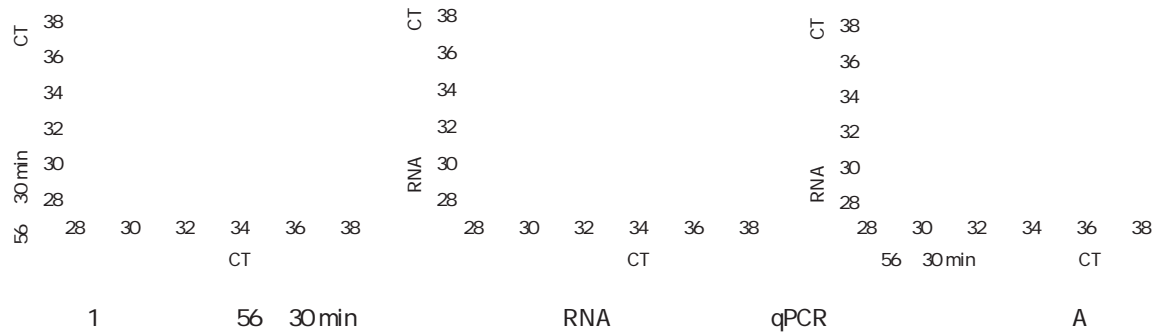


Figure 1 Correlation analysis of qPCR results among the un inactivated group, the inactivated group heated at 56 for 30 minutes and the guanidine inactivation group using detection kit A

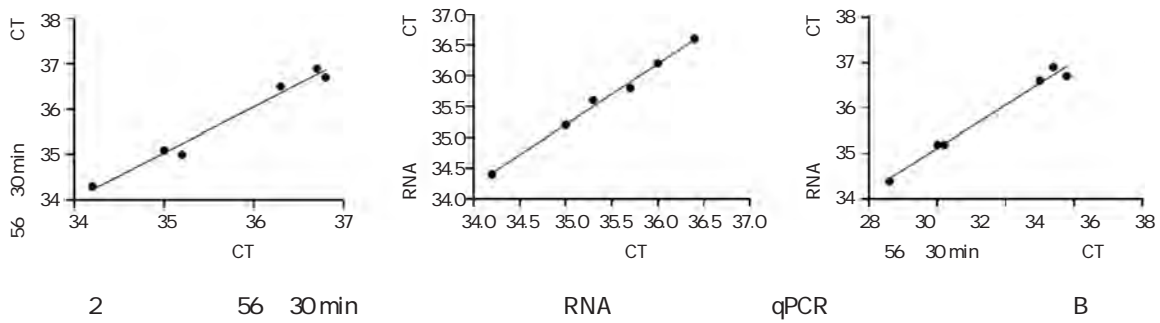


Figure 1 Correlation analysis of qPCR results among the un inactivated group, the inactivated group heated at 56 for 30 minutes and the guanidine inactivation group using detection kit B

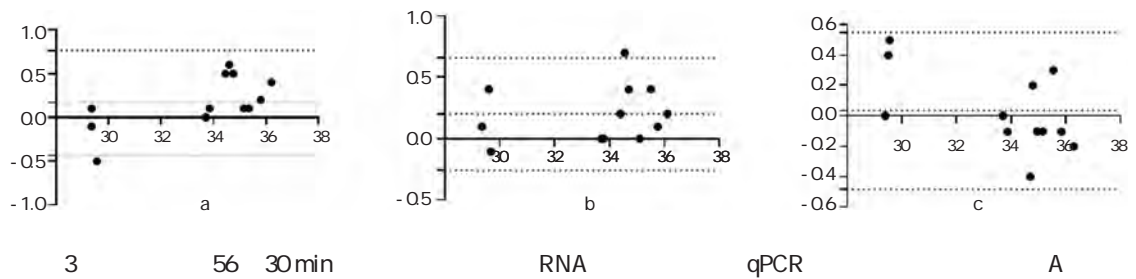


Figure 3 Consistency analysis of qPCR results among the un inactivated group, the inactivated group heated at 56 for 30 minutes and the guanidine inactivated group using detection kit A

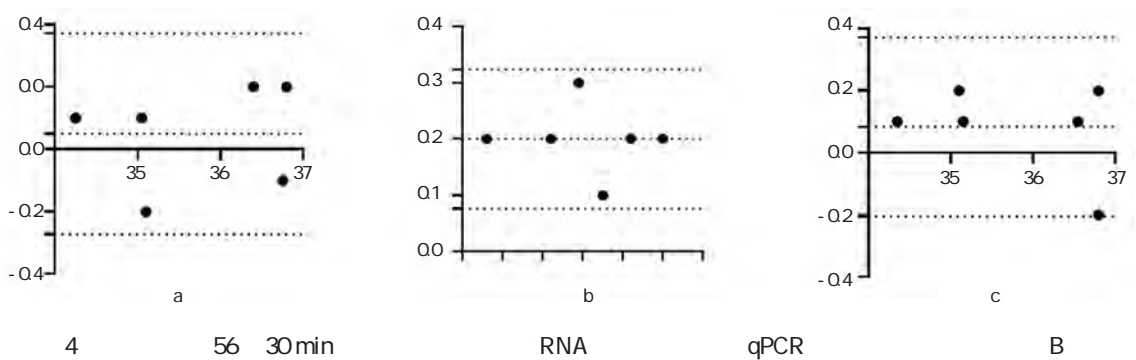


Figure 4 Consistency analysis of qPCR results among the un inactivated group, the inactivated group heated at 56 for 30 minutes and the guanidine inactivated group using detection kit B

56 30 min RNA
qPCR
2.3

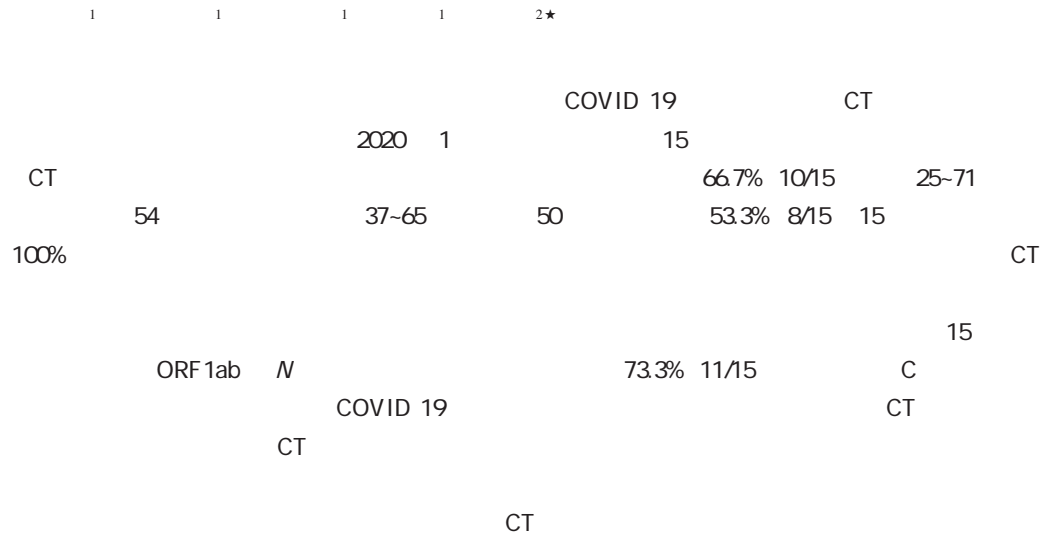
A B CT
=1.031 5 Bland Altman
qPCR
E •

• COVID 19

15

COVID 19

CT



Analysis of the clinical manifestations and computed tomography characteristics of patients with coronavirus disease-2019 COVID-19

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ABSTRACT Objective To analyze the clinical presentations and computed tomography characteristics of coronavirus disease 2019 COVID 19 and to further improve the understanding of the disease. Methods A retrospective analysis and summary of the clinical manifestations and CT imaging features were performed based on 15 patients with COVID 19 diagnosed in our hospital since January 2020. Results Most patients diagnosed in this study were men 67% 10/15 aged 25 to 71 years with a median age of 54 years in tertile range of 37 to 65 . Most of them were over 50 years old 53.3% 8/15 . All 15 cases had a history of contact with the affected area 100% . The clinical manifestations are fever cough dry cough or sputum with white mucus some accompanied by chest tightness shortness of breath and fatigue. Most patients were in early or advanced disease stages according to the chest imaging manifestations. They have a ground glass patchy shadow in bilateral pulmonary multi lobular lesions predominantly in the subpleural regions blurred lesion margins. They were also accompanied by thickening of blood vessels thickening of the pleura bronchial inflation and increased lymph nodes in the mediastinum in some cases. 15 patients have positive nucleic acid tests for the

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2. 572000

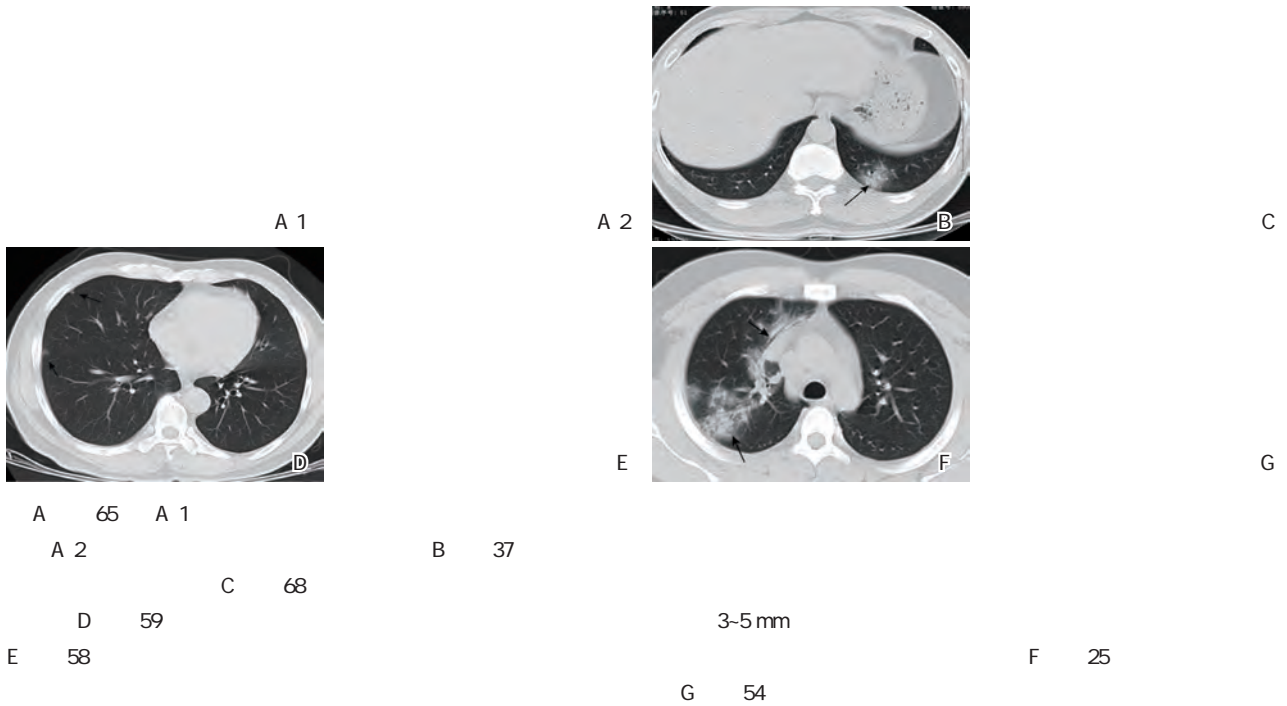
* E-mail zanmon@163.com

novel coronavirus ORF1ab and N genes. In addition 93% 14/15 of patients have high level of hypersensitivity C reactive protein. Conclusion COVID 19 patients have fever and cough at the early stage and there are abnormal chest CT examinations. The combination of epidemiological investigations clinical manifestations chest CT imaging characteristics analysis and nucleic acid detection and other laboratory test results will help early screening prevention and control of COVID 19.

KEY WORDS Coronavirus disease 2019 Clinical manifestations Computed tomography characteristics

2019 12
 1.2
 PCR 1.2.1
 β Sarbecovirus T310 04/T320 04
 orthocoronavirinae¹ 5 mL
 2019 novel coronavirus 2019 DA0633 2019 nCoV
 nCoV 2 se
 vere acute respiratory syndrome 2 SARS CoV 2
 WHO DA0930
 coronavirus disease 2019 COVID 19 DA3200
 ABI 7500
 2019 nCoV BC 5390
 M 53D
 4 19 84 237 LC M 5LEO I M 5LEO II M 53LH
 77 895 4 642 M 53P C CRP
 / COVID 19 7600 020 CT GE Light
² WHO 2020 2 1 COVID 19 Speed 16
³ 1.2.2
 ①
 ②
 15 EDTA K₂ 3 mL
 CT
 1 1.2.3
 1.1 56 30 min
 1 mL
 2020 1 25
 15 COVID 19 2019
 RT PCR nCoV
 15 1.2.4
 CT GE LightSpeed 16 CT

CT	120 kV	2.2			
200 mA	1.375 1	5 mm	7	8	62.5% 5/
5 mm	512x512		8	25% 2/8	
	L 500HU W 1 500		12.5% 1/8		
HU	L 50HU W 300HU		66.7% 10/15		
1.2.5			13.3% 2/15	86.7% 13/15	
	C	2.3 CT			
1.3		1			14
	SPSS 20.0				9
	$\bar{x} \pm s$	64.3%			2
M	IQR	14.3%	3	21.4%	
n %			1		
2		6	42.9%		
2.1		3	21.4%	1	7.1%
		4	28.6%		8
	66.7% 10/15	33.3% 5/15	57.1%		
	25-71	54			
	37-65	50			
30-40	33.3% 5/15	20-30	13.4%	1	2
2/15	15				1
100%					



1 COVID 19 CT
 Figure 1 The chest CT imaging of partial COVID 19 patients

2.4

ORF1ab N 4.5% 69.9%
 33.3% 5/15 25.5% 25.2%
 13.3% 2/15 13.3% 2/15 15
 73.3% 11/15 C 73.3% C
 60.0% 9/15

3

COVID 19
 2019 nCoV MERS CoV SARS CoV
 nCoV SARS CoV 2019
 80%^{1 4} SARS CoV 75% ~
 86.9%¹ SARS CoV 16 CT
 COVID 19
 2019 nCoV 99%
 2019 nCoV COVID 19
 5 6 CT
 2019 nCoV SARS CoV
 2 ACE2
 ACE2
 7 8
 86 2019 nCoV
 9
 COVID 19

10 11 31 44 672
 COVID 19 30-
 79

7.3¹² 30-71
 86.7% 2 1

SARS
 SARS
 COVID 19¹³ MERS 35%
 50 66.6%

MERS 14
 66.7% 10/15

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HCV RNA

1 2 1 1 1 1*

10 HCV RNA 1 005 2019 1
 PCR HCV HCV RNA HCV CMIA
 HCV RNA HCV S/CO
 1 005 HCV 228 22.7% HCV
 HCV RNA HCV PCR HCV
 RNA 83 36.4% HCV S/CO HCV RNA
 HCV S/CO ≥ 10.7 92.59%
 77.93% HCV HCV RNA

HCV RNA

Application of high-sensitivity HCV RNA detection in preoperative screening

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ABSTRACT Objective To investigate the application of high sensitivity HCV RNA detection in preoperative screening. Methods A total of 1 005 serum samples were collected from the preoperative screening patients in the Hospital Sun Yat sen University from January to October 2019. HCV antibodies and HCV RNA were detected using chemiluminescence microparticle immunoassay CMIA and fluorescence quantitative polymerase chain reaction FQ PCR . The comparison and correlation analysis between the S/CO value of HCV antibody and the high sensitivity HCV RNA test results were performed. The predictive efficacy of HCV antibody S/CO value for hepatitis C viremia was evaluated. Results Among the 1 005 serum samples 228 were positive for HCV antibody and the positive rate was 22.7%. HCV RNA were not detected from HCV antibody negative specimens. High sensitivity FQ PCR method detected 83 HCV RNA positive cases and the positive rate was 36.4%. There was no correlation between the HCV antibody S/CO value and the HCV RNA quantitative value. Using the critical S/CO value ≥ 10.7 with a sensitivity and specificity of 92.59% and 77.93% respectively can reduce the false positive rate for predicting hepatitis C viremia. Conclusion High sensitivity HCV RNA quantitative detection is applied for the preoperative screening of patients with hepatitis C which can accurately reflect the virus replication and infectivity in patients. Compared with the

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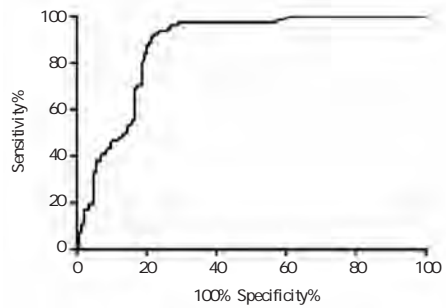
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traditional preoperative screening method. FQ-PCR can distinguish the current infection from the previous infection and has the advantages of rapid

HCV RNA 1
 HCV RNA HCV CMIA
 HCV HCV
 100% 78.3%
 36.4% 100% 85.6%
 2.2 HCV S/CO HCV RNA
 HCV S/CO
 1 S/CO
 S/CO HCV RNA
 1 Y=0.06
 617X + 4.259 Spearman r²=0.002 P=
 0.69 HCV S/CO HCV RNA

cut off
 Youden s index 0.705 2
 92.59% 77.93% S/CO ≥1
 HCV 228 HCV
 HCV RNA 36.4% 83/228 2



2 HCV S/CO
 ROC

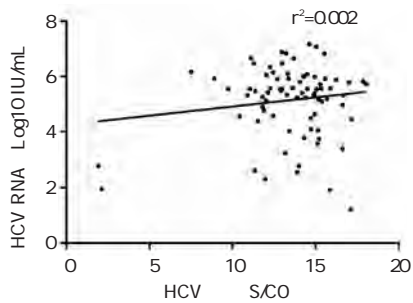
Figure 2 ROC curve of HCV antibody S/CO value for predicting hepatitis C viremia

1 HCV S/CO HCV RNA
 n %

Table 1 Distribution of HCV antibody S/CO value and high sensitivity HCV RNA detection results n %

S/CO	n	HCV RNA
4>S/CO≥1	87	1 1.1 86 98.8
8>S/CO>4	21	2 9.5 19 90.5
12>S/CO>8	29	15 51.7 14 48.3
16>S/CO>12	79	56 70.9 23 29.1
S/CO>16	12	9 75.0 3 25.0
	228	83 36.4 145 63.6

3



1 HCV S/CO HCV RNA

Figure 1 Scatter plot of HCV antibody S/CO value against high sensitivity HCV RNA quantitative detection results

2.3 HCV S/CO

HCV S/CO ROC
 area under the curve AUC 0.872 4
 ROC S/CO ≥10.7 HCV

HCV

HCV

HCV HCV 4
 HCV 2

HCV 5 HCV

PCR

PCR

Baleriola C 6 - 20
 - 70 HCV RNA - 20
 1.2 - 70 9 HCV RNA

- 80 1 HCV RNA
 HCV RNA 228 HCV
 36.4%
 HCV HCV

HCV RNA

HCV RNA
S/CO HCV RNA
S/CO HCV RNA
Spearman
HCV RNA
HCV
S/CO cut
off 10.7
S/CO ≥ 1
HCV HCV RNA
HCV
Pan J Kao HH Ranjbar Kermani F 79
HCV
S/CO ≥ 1 cut
off HCV HCV
RNA HCV HCV RNA
HCV HCV
10
HCV RNA 15 IU/mL
HCV
11
Ha J 12 HCV RNA 32 127
Architect anti HCV HCV
8 0.02% 8/32 127 HCV
RNA 13 148 590 ELISA
HCV HCV RNA 298
0.2% 14 307 440
ELISA HCV
1 HCV RNA 0.000 325%
HCV
15 HCV HCV RNA
HCV RNA
HCV HCV

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2 Glu504Lys 2

1* 2 1 2 1 1 1

2 T2DM 2 ALDH2 Glu504Lys

ALDH2 504 HbA_{1c}

2016 4 2017 12 629

402 2 227 ALDH2

ALDH2 HbA_{1c} 2 GL LL

37.9% 7.0% 30.8% 3.2% P<0.05 629

GG 390 GL 210 LL 29 ALDH2 2

GG 32.1% 125/390 GL 41.0% 86/210 LL

55.2% 16/29 ALDH2 2 OR =1.578 95% 1.131~

2.202 ALDH2 HbA_{1c} P>0.05 ALDH2 ALDH2

P>0.05 2 ALDH2 HOMA 1 IR

QUICKI ISI P>0.05 2

ALDH2 504 Glu504Lys 2

HbA_{1c}

2 2

Distribution characteristics of Glu504Lys polymorphism of acetaldehyde dehydrogenase 2 gene in Type 2 Diabetes Mellitus in Han Nationality

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ABSTRACT Objective To analyze the distribution characteristics of the Glu504Lys polymorphism of the aldehyde dehydrogenase 2 (ALDH2) gene in people with type 2 diabetes (T2DM) and to explore the relationship between ALDH2 gene mutation at position 504 and fasting blood glucose (HbA_{1c}) level and insulin resistance. Methods A retrospective study was conducted in 629 cases of physical examination and hospitalization in Foshan Traditional Chinese Medicine Hospital from April to December in 2016. There were 402 healthy controls and 227 patients with type 2 diabetes mellitus. The distribution of ALDH2 genotypes in the two groups was analyzed and the distribution levels of HbA_{1c}, fasting blood glucose and insulin resistance in ALDH2 genotypes of the two groups were compared. Results The proportions of GL and LL mutation genotypes in type 2 diabetes group were 37.9% and 7.0% respectively which were significantly higher than those in the physical examination control group (30.8% and 3.2% P<0.05). Among 629 Han subjects 390

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cases are GG 210GL and 29LL. With the increase in the number of ALDH2 gene mutations the risk of type 2 diabetes increases. The specific data are 32.1% 125/390 without mutation GG type 41.0% 86/210 with heterozygous mutation GL type and homozygous mutation LL Type 55.2% 16/29 . ALDH2 gene mutation is a risk factor for type 2 diabetes OR value = 1.578 95% confidence interval is 1.131-2.202 ALDH2 gene mutation has no effect on the distribution of HbA 1C level P>0.05 . There was no difference in the distribution of ALDH2 genotypes between men and women in the 2 groups P>0.05 . There was no significant difference in the distribution of insulin resistance index HOMA1 IR quantitative insulin sensitivity test index QUICKI and fasting insulin sensitivity index ISI of each type of ALDH2 genotype in type 2 diabetes group P>0.05 . Conclusion The proportion of ALDH2 gene mutation at position 504 in Han type 2 diabetes population is significantly higher than that in normal population. Glu504Lys mutation is a risk factor for type 2 diabetes but it was not significantly related to fasting blood glucose HbA 1C and insulin resistance.

KEY WORDS Aldehyde dehydrogenase 2 Type 2 diabetes mellitus Genotype Polymorphism genetic

2 acetaldehyde dehydrogenase 2 154 73 18-90
 ALDH2 57.78±15.30
 19 402 330
 72 17-87 38.63±9.47
 ALDH2 12q24 G→A ALDH2
 rs671 504 ALDH2
 glutamic acid G lysine L ALDH2
 G L GG GL 1.1.2 P>0.05
 LL ALDH2 2 5 1999 WHO
 ALDH2 Glu504Lys 5
 12 2 ≥11.1 mmol/L ≥7.0 mmol/L 75
 ALDH2 2 ≥11.1 mmol/L
 HbA_{1c} 20-80 2
 3 2 ALDH2 2
 Glu504Lys 47.80%
 22.0% 19.38% 4
 2
 1.2
 ALDH2 ABI 7500
 2 ALDH2 BR 526 24 ALDH2
 Glu504Lys ALDH2 G7
 HbA_{1c} TOSOH
 AU5400
 1 Centaur XP
 1.1
 1.1.1
 2016 4 2017 12 1.3
 2 227 2 8 3-4

mL ALDH2 ③ ALDH2
 EDTA 2Na BR 526 24
 2-
 8 3 1.5
 HOMA1 IR SPSS 22.0
 HOMA1 IR HOMA1 IR= × 1 Sample K S Kolmogorov Smirnov
 /22.5 ISI ISI=1/ $\bar{x} \pm s$
 × QUICK χ^2 P<0.05
 QUICKI ⑥
 1.4 ALDH2
 DNA ①DNA 2
 2 mL 2.1 T2DM ALDH2
 DNA ALDH2
 ②DNA ALDH2 P<0.01 2
 ABI 7500 1

Table 1 Distribution of ALDH2 genotypes in control group and T2DM group n %

	1	T2DM	ALDH2	n %
	GG	GL	LL	
T2DM	402 227 629	265 65.9 125 55.1 390 62.0	124 30.8 86 37.9 210 33.4	13 3.2 16 7.0 29 4.6
χ^2			9.489	7.255
P			0.009	0.007

2.2 ALDH2 2 1.578 95% 1.131~2.202
 2
 ALDH2 2.3 ALDH2
 2 ALDH2
 P<0.01 P>0.05 2
 L 2 ALDH2
 ALDH2 Glu504Lys L P>0.05
 2 P<0.01 3
 2 629 ALDH2 2 n %

Table 2 Proportion of type 2 diabetes mellitus in 629 subjects with ALDH2 genotype n %

	GG	GL	LL	n %
T2DM	227	125 32.1	86 41.0	16 55.2
DM	402	265 67.9	124 59.0	13 44.8
	629	390	210	29
χ^2			9.256	7.255
P			0.002	0.007

3 ALDH2 n %
 Table 3 Distribution of ALDH2 genotypes between male and female n %

		3			ALDH2			n %	
		GG	GL	LL	GG	GL	LL	n	%
		330	221 67.0	100 30.3	9 2.7	221 67.0	109 33.0		
		72	44 61.1	24 33.3	4 5.6	44 61.1	28 38.9		
	χ^2			1.946			0.903		
	P			0.378			0.342		
T2DM		154	85 55.2	58 37.7	11 7.1	85 55.2	69 44.8		
		73	40 54.8	28 38.4	5 6.8	40 54.8	33 45.2		
	χ^2			0.014			0.003		
	P			0.993			0.955		

2.4 2

$\chi^2=0.606 P>0.05$

2.4.1 HbA_{1c}

HbA_{1c} Kolmogorov Smirnov

Z =-1.393 $\chi^2=0.041 P<0.05$

Z =-0.763

GG GL LL

HbA_{1c}

F =0.291 P=0.748 P>0.05

GG

GL+LL HbA_{1c}

F =0.546 P=0.461 P>0.05

4

4 T2DM

$\bar{x} \pm s$

Table 4 The distribution level of each biochemical serological index in each genotype $\bar{x} \pm s$

		GG	GL	LL	$\bar{x} \pm s$	
HbA _{1c}		8.818±2.559	9.023±2.296	8.955±2.788	8.818±2.559	9.014±2.350
	mmol/L	8.897±3.546	9.851±4.431	6.946±2.424	8.897±3.546	9.453±4.321
		F =3.690 P=0.027			F =1.035 P=0.310	
mU/L		19.46±16.59	20.57±17.65	17.82±11.31	19.46±16.59	20.15±16.83
		F =-0.205 P=0.815				
		7.553±6.979	9.399±9.463	5.392±3.713	7.553±6.979	8.846±8.986
		F =1.949 P=0.145				
		0.302±0.032	0.299±0.035	0.311±0.034	0.302±0.032	0.301±0.035
		F =-0.865 P=0.423				
		6.798±6.281	8.459±8.517	4.853±3.341	6.798±6.281	7.9613±8.088
		F =1.949 P=0.145				

2.4.2

5 T2DM

Table 5 The statistical results of FBG levels among genotypes in the T2DM group

		F =3.690 $\chi^2=$	
0.027 P<0.05	GL	9.851	GG
8.897	LL	6.946	
GG	GL		
	LL	GG	
	GL		
GL+LL			
5			

		F	I J	P
GG VS		3.690	/	/ 0.027
GL+LL		1.035	/	/ 0.310
GG VS	GL	/	-0.954	5.626 0.092
GG VS	LL	/	1.951	1.113 0.087
GL VS	LL	/	2.904	1.156 0.013

3

ALDH2
30%~50%
15%~30%⁷
ALDH2 34.1%
⁸ 2
44.9% ALDH2
2
ALDH2 Glu504Lys 2
ALDH2

DNA

¹⁸
ALDH2 Glu504Lys
2
2
9

HbA_{1c} 8-12

ALDH2 HbA_{1c}
¹³ ALDH2
2 HbA_{1c}

ALDH2 3 >

1UÂy•2

6

	1	2	1	1	1	1	1*	1*
			27	6				
			6				PCR	
DNA						27	6	
	14					6		
							6	
		6						

Establishment of immortalized lymphocyte cell lines with mutation of the glucose -6- phosphate dehydrogenase gene

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ABSTRACT Objective To establish immortalized lymphocyte cell lines by collecting 27 clinical peripheral blood samples harboring glucose 6 phosphate dehydrogenase G6PD gene mutations. Methods The glucose 6 phosphate dehydrogenase gene mutation detection kit fluorescence PCR melting curve method was used to screen the mutation type of clinical samples and then peripheral blood was collected for transformation of immortalized lymphocyte lines. The genomic DNA of the successfully established lymphocyte line was extracted and verified by the next generation sequencing method. Results We established 27 immortalized lymphocyte cell lines including 14 different homozygous or heterozygous G6PD mutation types. The genetic mutation types of immortalized lymphocyte lines were the same as those originally indicated in clinical samples. Conclusion The established immortalized lymphocyte cell lines with G6PD mutations in this study have stable genetic information and can be used as a national reference resource to evaluate the performance of the G6PD gene mutation kit

KEY WORDS Glucose 6 phosphate dehydrogenase Gene mutation Immortalized cell line

2016YFC1000300

- 1. 100050
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6 glucose 6 phosphate de PCR
hydrogenase G6PD X DNA
2 8 Xq28 G6PD G6PD

1 2 SLAN 96S PCR
MGISEQ
2000
G6PD
G6PD G6PD X 1.2
1.2.1
6
PCR
1.2.2
1.2.2.1 EBV B95 8
RPMI 1640 FBS
2-3
10
3 2 000 rpm 37
0.2 m μ 10 min
6
6
1.2.2.2 B
5 mL
RPMI 1640
6 2 000 rpm 20 min
8 mL 2 1 500 rpm RPM1640
EBV 10 min
1.5 mL
1 A 0.4 mL T25
1.1 37 5% CO₂
1.1.1 1.2.2.3 24-
48 h
10 mL 33
1.1.2 1 300 rpm 5
RPMI 1640 FBS 3-6×10⁶ /mL
GIBCO A DMSO 1 300 rpm 5
10% DMSO
6

A B C
 A O B 15 C 30
 2 O 15 30 ×50

Figure 2 The result of immortalized lymphocyte cell line transformation on day 0 day15 and day 30 ×50

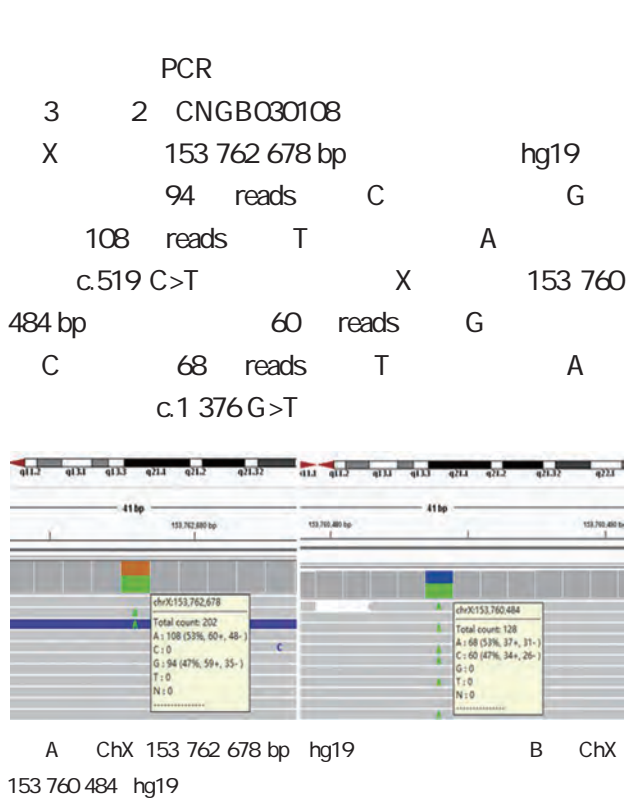


Figure 3 The sequencing results of CNGB030108

3

EB EBV
 B
 A T
 T B

2

Table 2 The verification results of immortalized lymphocyte cell lines

1	CNGB030075	c.392G>T
2	CNGB030078	c.871G>A
3	CNGB030079	c.871G>A
4	CNGB030081	c.95A>G
5	CNGB030082	c.95A>G
6	CNGB030084	c.95A>G
7	CNGB030085	c.1024C>T ; c.1388G>A
8	CNGB030086	c.1388G>A
9	CNGB030101	c.1388G>A
10	CNGB030102	c.1376G>T
11	CNGB030103	c.871G>A
12	CNGB030104	c.1388G>A ;
13	CNGB030105	c.1388G>A
14	CNGB030106	c.392G>T
15	CNGB030107	c.1376G>T
16	CNGB030108	c.1376G>T ; c.519C>T
17	CNGB030109	c.95A>G
18	CNGB030110	c.1360C>T
19	CNGB030111	c.1388G>A
20	CNGB030112	c.1388G>A ; c.519C>T
21	CNGB030113	c.1024C>T
22	CNGB030114	c.95A>G
23	CNGB030115	c.1376G>T
24	CNGB030116	c.95A>G
25	CNGB030117	c.1388G>A
26	CNGB030119	c.392G>T
27	CNGB030120	c.1388G>A ; c.392G>T



		81370615	
1.	ICU		450000
2.			450000

*

increased inflammatory factors. It is also a risk factor for the prognosis of patients with sepsis and cardiac dysfunction.

KEY WORDS Sepsis cardiac function miR 21 miR 155 Prognosis

^{1 2} p 55

2 P>0.05 CRP PCT cTnl BNP
 LVEF<50% <LVEF≥50% <
 2.1 3 P<0.05 1
 3 BMI

Table 1 comparison of general data among 3 groups $\bar{x} \pm s$

	n=40	LVEF≥50%	n=38	LVEF<50%	n=20	Z/F	P
/	23/17	22/16		12/8		0.036	0.982
BMI kg/m ²	42.39±9.59	45.12±10.92		40.39±11.87		1.432	0.244
CRP mg/L	22.93±5.82	23.14±6.14		22.46±5.67		0.087	0.917
PCT ng/mL	3.28±0.62	30.38±7.82		61.39±9.94		530.616	0.000
cTnl ng/mL	0.58±0.08	3.29±0.68		14.69±3.94		417.803	0.000
BNP ng/L	0.45±0.08	0.68±0.12		1.52±0.20		481.609	0.000
	32.93±7.95	63.29±10.29		132.29±21.29		417.546	0.000

2.2 3 miR 21 miR 155 IL 2 ICAM 1 LVEF<50%
 LVEF≥50%
 3 miR 21 miR 155 TNF α P<0.05 2

Table 2 comparison of circulation miR 21 miR 155 and serum TNF α IL 2 ICAM 1 among 3 groups $\bar{x} \pm s$

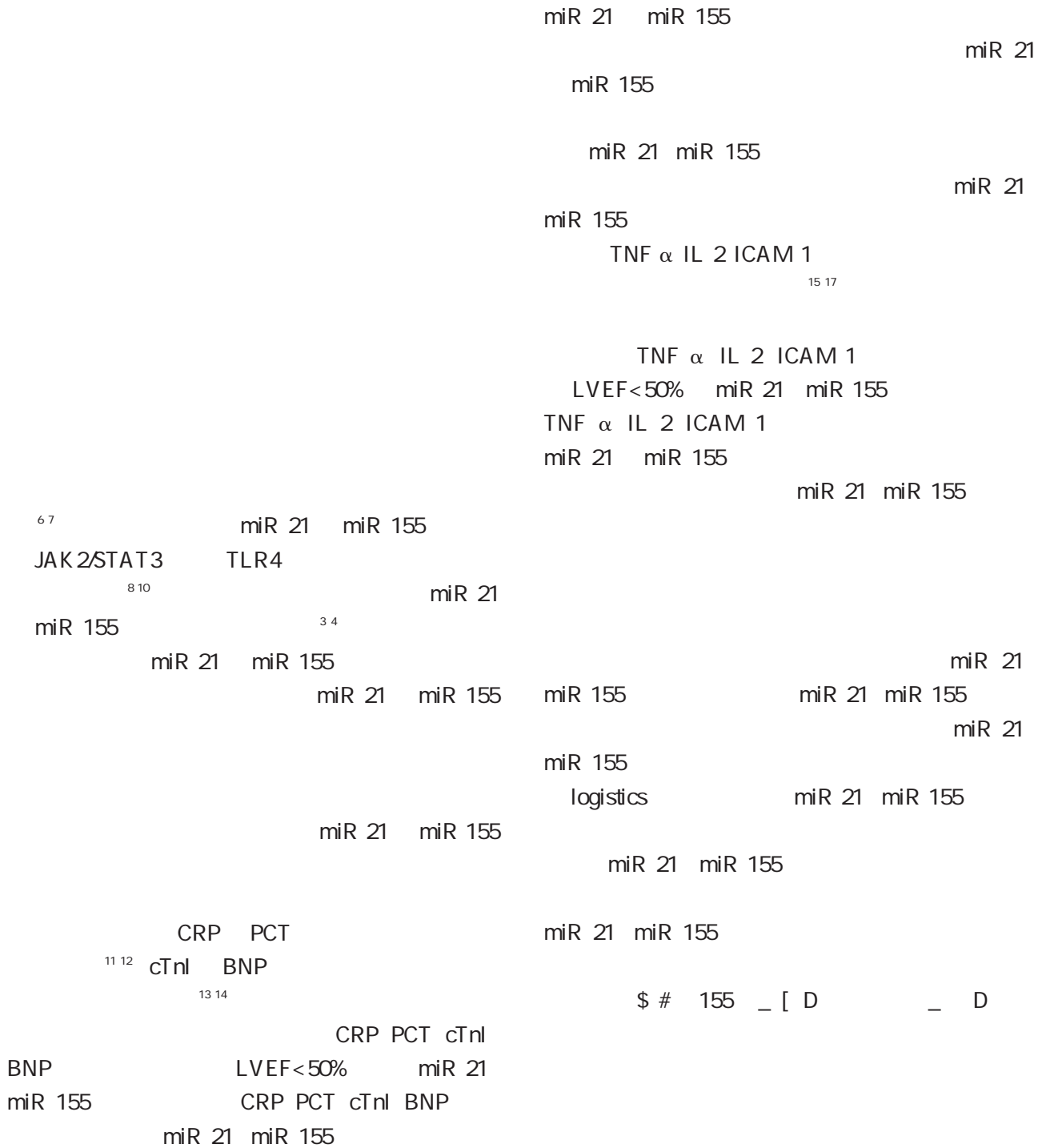
	n	miR 21	miR 155	TNF α ng/L	IL 2 ng/L	ICAM 1 ng/mL
	40	1.02±0.19	0.97±0.16	9.38±1.32	14.28±2.37	126.49±17.96
LVEF≥50%	38	1.42±0.23	1.38±0.19	20.19±4.82	32.93±6.48	349.58±62.39
LVEF<50%	20	2.19±0.42	1.83±0.32	57.92±9.94	82.93±13.28	649.39±85.93
F		129.091	113.378	543.380	586.608	592.120
P		0.000	0.000	0.000	0.000	0.000

2.3 LVEF<50% miR 21 miR 155 2.4 LVEF<50%
 LVEF<50% miR 21 miR 155 LVEF<50% miR 21
 CRP PCT cTnl BNP TNF α IL 2 miR 155 TNF α IL 2 ICAM 1
 ICAM 1 P< P<
 0.05 3 0.05 4

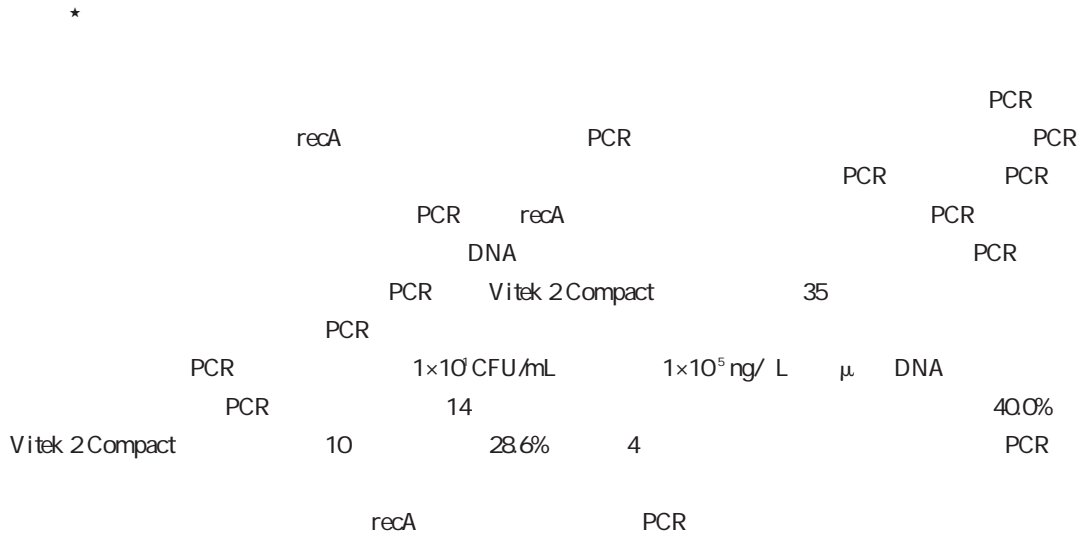
Table 3 correlation between miR 21 miR 155 and serum index in LVEF <50% group

	miR 21		miR 155	
	r	P	r	P
CRP	0.273	<0.05	0.410	<0.05
PCT	0.306	<0.05	0.377	<0.05
cTnl	0.227	<0.05	0.342	<0.05
BNP	0.285	<0.05	0.266	<0.05
TNF α	0.329	<0.05	0.325	<0.05
IL 2	0.268	<0.05	0.367	<0.05
ICAM 1	0.402	<0.05	0.291	<0.05

2.5 LVEF<50%
 LVEF<50%
 miR 21 miR 155 CRP
 PCT cTnl BNP TNF α IL 2 ICAM 1
 logistics miR 21 miR 155
 PCT TNF α IL 2 ICAM 1
 LVEF<50%
 CRP BNP LVEF<50%
 5
 3



PCR



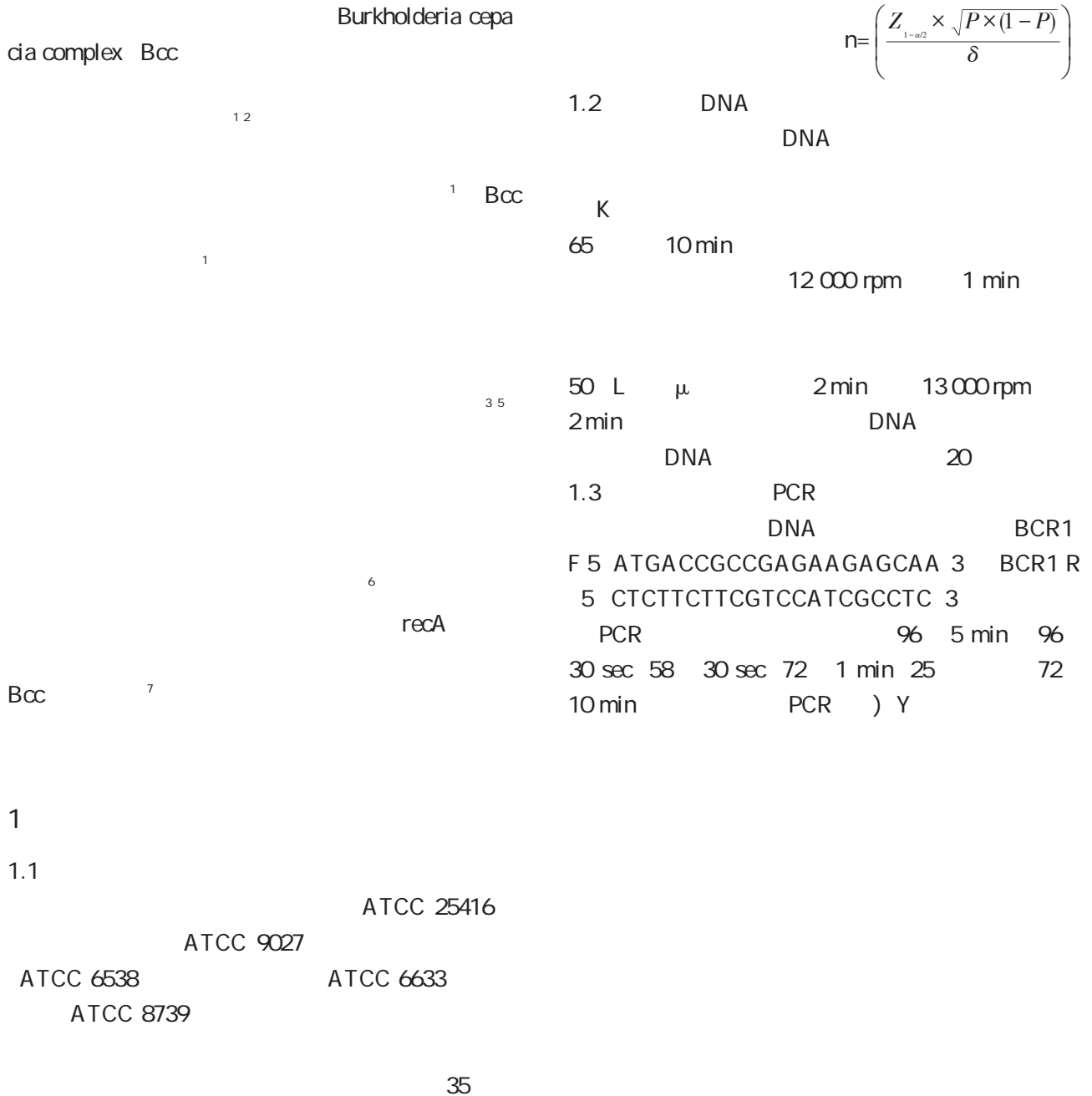
Establishment of nested fluorescent quantitative PCR for rapid detection of

Burkholderia cepacia

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cating that the nested fluorescent quantitative PCR had more accurate detection performance. Conclusion Nested fluorescent quantitative PCR is an effective rapid and direct method for clinical detection of Burkholderia cepacia.

KEY WORDS Burkholderia cepacia RecA gene Nested fluorescent quantitative PCR Clinical detection



$Z_{1-\alpha/2}=1.96$ $P=0.9$

$\delta=0.1$
35

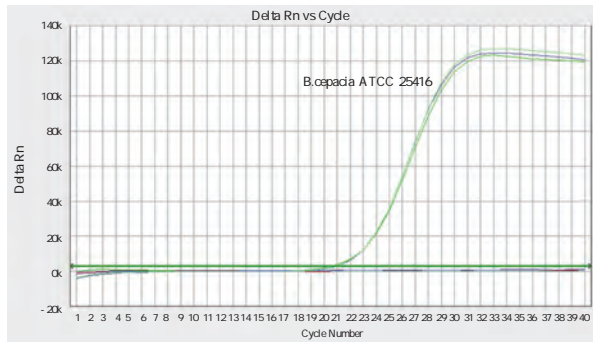
recA

PCR

B.cepacia

ATCC 25416

1



1

PCR

Figure 1 Amplification curves of nested quantitative PCR for different bacterial strains

2.2 PCR

1×10^5 1×10^4 1×10^3 1×10^2

1×10^1 1×10^0 CFU/mL

PCR

1×10^0 CFU/mL

1×10^0 CFU/mL

2

DNA

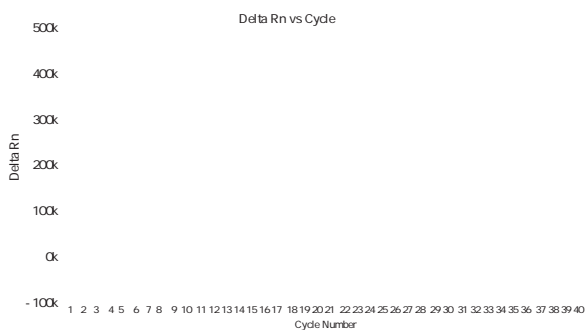
1×10^1 1×10^2 $1 \times$

10^3 1×10^4 1×10^5 1×10^6 ng/ L μ 1 L μ

1×10^6 ng/ L μ 1 \times

10^5 ng/ L μ

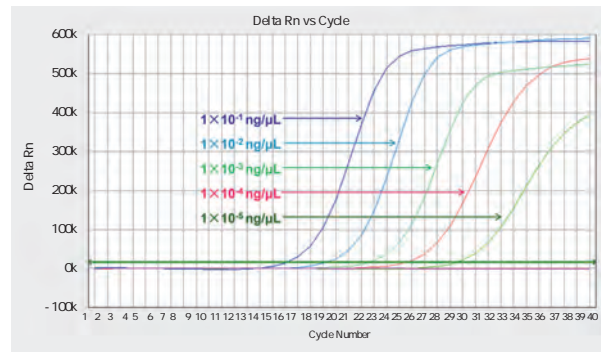
3



2

PCR

Figure 2 Amplification curves of Burkholderia cepacia with different concentrations by nested quantitative PCR



3

DNA

PCR

Figure 3 Amplification curves of Burkholderia cepacia genomic DNA in different concentrations by nested quantitative PCR

2.3

PCR

35

PCR Vitek 2 Compact

Vitek 2 Compact

10

4

Vitek 2 Compact

4

14

Vitek 2 Compact

Compact

PCR

Vitek 2

$\chi^2=0.560$ $P=0.454$

3

16S rRNA

8

16S rRNA

98%~100%

Bcc

Bcc

8

recA

Bcc

8 10

recA

8

recA

Bcc

recA
 Bcc
 98.9% recA
 98.9%
 100%
 recA
 11 12
 Bcc
 recA
 recA
 recA

Jimenez ²⁰
 PCR 16S rRNA
 10 fg/ L μ 0.98
 Wright ²¹ recA PCR
 5 D

- { 1 \$ # CE6* •• !
 WD

gyrB
 13 hisA
 442 bp hisA DNA
 hisA Bcc
 Bcc ¹⁴
 /

15 MALDI TOF MS
 Furlan ¹⁶
 Bcc
 VITEK 2 MALDI TOF MS
 16S rRNA recA
 PCR Bcc recA PCR
 Lambiase ¹⁷
 MALDI TOF MS Bcc
 Zakharova ¹⁸ β
 PCR

Bcc
 β
 D β
 Bcc
 PCR

19 III
 PCR
 10 copies/ L μ



results of influenza A and B virus in oropharyngeal swab samples.

KEY WORDS Virus inactivation Quantitative real time polymerase chain reaction Influenza viruses Oropharyngeal swab

1 RNA

1.3 -20 1 mL
200 L μ

1.2 56

30 min

1.4 PCR

severe acute respiratory syndrome coronavirus 2 SARS CoV 2

RNA ABI7500 PCR

50 15 min → 95 15 min →

94 15 s → 58 45 s 45

2011 cycling threshold CT

56 30 min PCR

67

56 30 min 1.5

① FAM

quantitative real time polymerase chain reaction qPCR

Ct ≤39.7 FAM

Ct ≤38.0 ② FAM

Ct >39.7 FAM

Ct >38.0 FAM

1

1.1

2020 1 31 2020 2 15

47 qPCR

21 26

n=27 Ct ≤

26 n=16 26 Ct ≤33 n=4

33 < Ct ≤ 39

14

qPCR

1.2

ABI7500 PCR Thermofisher

qPCR

21

1.6

SPSS 26.0 GraphPad Prism

Version 8.0 $\bar{x} \pm s$

Bland Altman t

P < 0.05

2

2.1

qPCR

26

qPCR
 100% 56 30 min Ct
 P>0.05 1 2

1 qPCR

Table 1 The qPCR results of unheated and heat inactivation

Ct		qPCR			
32.52	32.83	0.31	30.36	28.72	-1.64
30.93	30.38	-0.55	25.60	26.73	1.13
30.26	36.64	6.38	28.31	25.58	-2.74
35.73	26.12	-9.61	23.02	23.81	0.79
26.55	20.79	-5.76	23.03	23.39	0.36
20.52	26.46	5.94	22.48	22.22	-0.26
27.74	31.06	3.33	20.80	21.67	0.87
24.05	23.32	-0.73	23.23	23.45	0.23
26.37	24.20	-2.18	20.88	21.46	0.58
28.18	27.05	-1.13	20.50	20.94	0.44
25.58	25.81	0.22	24.13	24.51	0.38
28.53	28.62	0.09	24.78	25.11	0.32
22.78	23.44	0.66	26.25	26.65	0.40
23.04	28.60	5.57	20.98	21.99	1.01
23.03	28.32	5.29	25.42	26.24	0.82
23.27	26.24	2.97	19.33	17.54	-1.79
19.56	19.10	-0.46	27.60	28.15	0.55
22.92	28.73	5.80	20.24	22.38	2.15
22.76	27.34	4.59	32.00	31.27	-0.73
37.46	38.17	0.71	26.59	25.15	-1.44
34.84	33.72	-1.13	26.11	26.66	0.55
			32.38	28.77	-3.60
			27.67	26.91	-0.76
			27.34	28.38	1.05
			27.64	28.26	0.62
			18.88	18.88	0

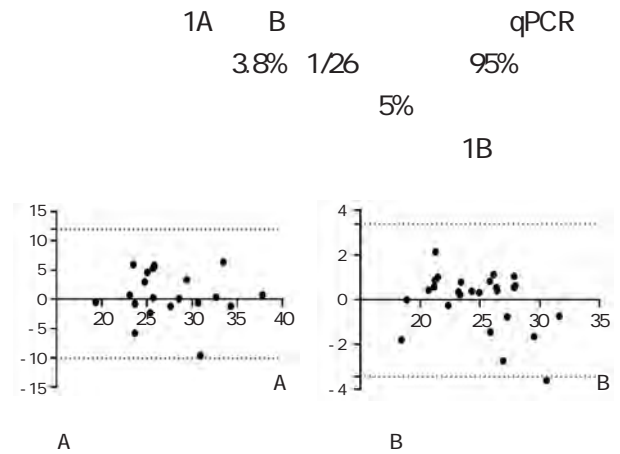
2 qPCR $\bar{x} \pm s$

Table 2 Comparison of the qPCR results between unheated and heat inactivation $\bar{x} \pm s$

n	t	P
Ct 21 26.98±5.07 27.95±4.76 -1.100	0.284	
Ct 26 24.83±3.77 24.80±3.32 0.108	0.915	

2.2 qPCR

qPCR 95%



1 Figure 1 A analysis of consistent results between unheated and heat inactivation

2.3

qPCR

14

qPCR

100%

3

8 9

10

11 12

13

SARS

56 60 min 56 30 min

SARS

30 min 60

min

14

56 30 min 75%

15

56
RNA
RNA

RNA ③
56 30 min

Ct 1
Ct 2
Ct 3
RNA ①
Ct 4

②

qPCR

③

16

qPCR

56 30 min
①

②

4

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2 UAER

hs CRP

	1	2*	1																	
				2	T2DM			CIMT	C											
hs CRP				UAER			2018	1	2019	12										
T2DM	63					CIMT	CIMT			CIMT≤0.9 mm	18									
		CIMT0.9-1.2 mm	23			CIMT>1.2 mm	22	3			T2DM									
BMI	WHR		SBP		DBP		TC		TG											
HDL C				LDL C		FPG		SCr		FINS										
HbA1c	2h	2hPG		C		hs CRP				UAER										
CIMT	hs CRP	UAER			3		BMI	WHR	SBP	DBP	FINS	SCr								
		P>0.05	3	HbA1c	TC	HDL C	LDL C	hs CRP	UAER											
P<0.05			FPG	2hPG	TG					P<0.05	T2DM									
CIMT	FPG	2hPG	HbA1c	TG	TC	LDL C	hs CRP	UAER		P<0.05	HDL C									
	P=0.027		T2DM	CIMT	hs CRP	UAER				hs CRP	UAER									
	T2DM			AS																
	2						C													

Correlation between carotid intima thickness and hs-CRP UAER in patients with type 2 diabetes mellitus

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ABSTRACT Objective To analyze the correlation between carotid medial intimal thickness CIMT and high sensitivity C reactive protein hs CRP and urine protein excretion rate UAER in patients with type 2 diabetes T2DM . Methods 63 T2DM patients admitted to our hospital from January 2018 to December 2019 were collected and the color Doppler ultrasound diagnostic instrument was used to detect CIMT. Based on CIMT grouping 18 patients in normal group CIMT ≤0.9 mm and 23 in thickened group CIMT 0.9-1.2 mm and 22 in plaque group CIMT>1.2 mm . The age sex T2DM course BMI WHR systolic blood pressure SBP diastolic blood pressure DBP total cholesterol TC triglyceride TG high density lipoprotein cholesterol HDL C Low density lipoprotein cholesterol LDL C fasting blood glucose FPG serum creatinine SCr fasting insulin FINS glycated hemoglobin HbA1c high sensitivity C reactive protein hs CRP and 2h blood glucose after meals 2hPG urinary protein excretion rate

2017

1704f0804042

1.

230011

2.

230011

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E-mail ahmusyy@sina.com

UAER were compared and the correlation between CIMT and hs CRP and UAER was analyzed. Results There were no statistically significant differences in age gender BMI WHR SBP DBP FINS and SCr in the three groups $P>0.05$. The differences in the course of disease HbA1c TC HDL C LDL C hs CRP and UAER in the three groups were statistically significant $P<0.05$. FPG 2hPG and TG in the plaque and thickening group were significantly higher than those in the normal group $P<0.05$. CIMT was positively correlated with the course of disease FPG 2hPG HbA1c TG TC LDL C hs CRP and UAER in T2DM patients $P<0.05$ and negatively correlated with HDL C $P=0.027$. Conclusion CIMT and hs CRP are closely related to UAER in T2DM patients. The combined detection of hs CRP and UAER can predict the early assessment of macrovascular disease or AS in T2DM patients.

KEY WORDS 2 diabetes mellitus Carotid intima thickness Hypersensitive C reactive protein Urinary protein excretion rate Correlation

2 Type 2 Diabetes Mellitus ⑧
 T2DM T2DM ⑨
 1
 T2DM A thero 1.2
 sclerosis AS 1.2.1 CIMT
 2 T2DM LOGIQ500
 Carotid Intima MediaThickness CIMT GE
 AS 3 8.2-100
 7
 4 T2DM
 AS C
 C reactive Protein CRP AS
 T2DM CIMT
 CRP C
 Highsensitivity C reactive protein hs CRP
 Urinary albumin excretion rate UAER
 T2DM CIMT hs CRP UAER

1

1.1

	2018	1	2019	12
T2DM	63		53-72	
	63.81±6.02	41	22	
	2		2017	
5	1-17		9.52±4.17	
	①T2DM	5 ②	3	
	③		④	
	⑤			⑥
				①
	②		③	
④		⑤	⑥	⑦

moglobin A 1c HbA 1c

C	Highsensitivity C reactive protein
hs CRP	2 h
2 mL	2 h 2hPostprandial blood glu
cose 2hPG	3 h

2.2 T2DM CIMT
 T2DM CIMT FPG 2hPG
 HbA1c TG TC LDL C hs CRP UAER
 P<0.05 HDL C
 P<0.05 2

2 T2DM CIMT
 Table 2 T2DM Correlation analysis between patient CIMT and other factors

	r	P
FPG	0.196	0.039
2hPG	0.264	0.015
HbA1c	0.253	0.019
TG	0.297	<0.001
TC	0.246	0.018
HDL C	0.255	0.015
LDL C	-0.249	0.027
FINS	0.316	0.008
SCr	0.197	0.302
hs CRP	0.062	0.575
UAER	0.329	<0.001
	0.288	<0.001

2.3 Logistic
 T2DM CIMT >1.2 mm
 FPG 2hPG HbA1c TG TC
 HDL C LDL C FINS SCr hs CRP UAER
 Logistic
 HbA1 TC LDL C hs CRP UAER T2DM
 P<0.05 3

3 Logistic
 Table 3 Risk factors Logistic regression analysis of carotid plaque

	β	Wald	OR	95%CI	P
HbA1c	2.778	4.982	1.546	1.029-2.142	0.025
TC	2.661	4.831	1.438	0.985-2.021	0.023
LDL C	0.762	4.125	1.223	1.006-1.372	0.029
hs CRP	0.613	3.686	1.825	1.297-2.605	0.035
UAER	3.079	6.407	28.464	2.145-73.212	<0.001
	2.136	4.356	8.564	1.208-43.597	<0.001

3

60%
 7
 8
 T2DM
 AS

AS
 T2DM
 9 T2DM AS

Jamshid 10
 CRP T2DM AS
 CRP T2DM
 AS
 CRP T2DM
 T2DM CIMT hs CRP
 T2DM AS
 12 T2DM
 13 CIMT 24 h
 UAER T2DM CIMT
 Logistic
 T2DM
 T2DM

AS 14
 UAER
 HbA1 TC LDL C T2DM
 T2DM
 CIMT 15

T2DM CIMT hs CRP
 UAER hs CRP UAER
 T2DM AS

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OPG TNF α

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GDM OPG TNF α /TNF α
 40 GDM

OPG TNF α OPG TNF α
 BMI TG TC LDL C P>0.05

FBG HDL C TG/TC TC/HDL C LDL C/HDL C AIP HOMA IR QUICKI
 P<0.05 GDM TNF α NGT OPG /TNF α r=-0.68

NGT P<0.05 /TNF α r=0.69 P<0.05

P<0.05 r=-0.39 P<0.05 r=0.69 P<0.05

/TNF α GDM OPG
 /TNF α GDM /TNF α
 GDM

α

Diagnostic analysis of serum OPG TNF - α and adiponectin in gestational diabetes mellitus

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ABSTRACT Objective To explore OPG TNF α adiponectin and adiponectin/TNF α ratio in the blood circulation of gestational diabetes mellitus (GDM) pregnant women and the relationship with metabolic syndrome biomarkers and clinical significance. Methods 40 women with GDM and 40 normal pregnant women were recruited in this study. The levels of serum OPG TNF α and total adiponectin were measured using enzyme linked immunosorbent assay (ELISA). The correlation between OPG TNF α and adiponectin and the degree of GDM was analyzed. Results There was no statistically significant difference in gestational age BMI TG TC LDL C P>0.05. The age of pregnant women FBG HDL C TG /TC TC /HDL C LDL C/HDL C AIP insulin HOMA IR and QUICKI were statistically significant between two groups P<0.05. The expression level of TNF α in the GDM group was higher than that in the control group. The ratio of OPG adiponectin and adiponectin /TNF α was significantly lower than that in the control group P<0.05. Adiponectin/TNF α ratio was negatively correlated with insulin resistance r=-0.68 P<0.05. Triglycerides r=-0.39 P<0.05 were positively correlated with insulin sensitivity r=0.69 P<0.05. Multiple linear

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regression analysis showed that the adiponectin/TNF α ratio was independently related to insulin resistance. Binary logistic regression analysis indicated that GDM was positively correlated with OPG levels and negatively correlated with adiponectin/TNF α ratio. Conclusion the OPG level in the GDM group increased while the adiponectin/TNF α ratio decreased significantly. This ratio may be an effective biomarker for assessing pregnant women's high risk of insulin resistance and dyslipidemia as well as GDM diagnostic and treatment monitoring targets.

KEY WORDS Gestational diabetes mellitus Osteoprotein Tumor necrosis factor α Adiponectin Insulin resistance

Gestational diabetes mellitus ② ③

GDM 1.2

GDM 4 GDM 7 100 100 g 250 mL

GDM GDM 30 4

osteoclastogenesis inhibi

tory factor OPG α nmol/L 5.6 nmol/L 10.3

TNF α adiponectin nmol/L 8.6 nmol/L

⁵ OPG 1.3 GDM

TNF α AMP 5 mL 3 000r/min 5 min

⁶ Thermo Multiskan FC

GDM OPG TNF α TNF α Olympus AU400

FBG TC

TG HDL C

<10

normal glucose tolerance LDL C Friedewald 8

NGT GDM OPG TC/HDL C LDL C/HDL C TG/

TNF α /TNF α TC log TG/HDL C AIP TINS ⁹

1 1.4 HOMA IR

1.1 HOMA IR= FPG \times FINS /225

2018 1 2018 12 10 QUICKI

GDM GDM 40 40 QUICKI=1/ logFINS U/mL μ +logFBG mg/dL

28 NGT 24- 1.5 ¹¹

SPSS 19.0

A /mL

① ②

22~35 ①

2
2.1
BMI TG TC LDL C
P>0.05 FBG
HDL C TG/TC TC/HDL C LDL C/HDL C AIP
HOMA IR QUICKI
P<0.05 1

Table 1 Biochemical characteristics of gestational diabetes mellitus GDM and normal pregnant women $\bar{x} \pm s$

	NGT n=40	GDM n=40	t	P
BMI kg/m ²	27.25±1.18	27.66±1.17	1.560	>0.05
FBG mg/dL	78.45±1.29	100.83±1.54	70.458	<0.001
TG mg/dL	221.83±74	256.12±69.12	2.142	>0.01
TC mg/dL	242.67±30.18	225.84±39.88	2.128	>0.01
HDL C mg/dL	52.21±2.95	34.7±2.71	27.646	<0.001
LDL C mg/dL	146.1±27.84	139.92±20.11	1.138	>0.05
TG/TC	0.94±0.06	1.17±0.1	12.474	<0.001
TC/HDL C	5.32±0.54	7.88±0.65	19.160	<0.001
LDL C/HDL C	3.23±0.36	5.17±0.58	17.974	<0.001
AIP	0.63±0.04	0.87±0.04	26.833	<0.001
IU/mL μ	9.54±0.55	12.56±0.9	18.109	<0.001
HOMA IR	1.85±0.11	3.11±0.22	32.398	<0.001
QUICKI	0.36±0.01	0.33±0.01	13.416	<0.001

2.2 NGT GDM OPG TNF α

GDM TNF α NGT
OPG /TNF α
NGT NGT HOMA IR
/TNF α TNF α
QUICKI /TNF α
TNF α LDL C/HDL C
TG/TC 2

Table 2 OPG TNF α and adiponectin levels of gestational diabetes mellitus GDM and normal pregnant women $\bar{x} \pm s$

	n	OPG	Adiponectin g/mL μ	TNF α pg/mL	Adiponectin/TNF α ratio
NGT	40	2224.76±881.47	6.37±0.59	115.68±12.64	4.80±0.07
GDM	40	1148.15±700.13	4.50±0.38	225.08±27.35	4.31±0.05
t		6.049	16.853	22.964	36.026
P		<0.001	<0.001	<0.001	<0.001

2.3 OPG

HOMA IR TNF α TG
/TNF α
QUICKI TNF α TG TG/TC
/TNF α
3
3

Table 3 Research and analysis of the correlation of clinical indicators

	HOMA IR		QUICKI	
	r	P	r	P
TNF α	0.38	0.001	-0.38	0.001
TG	0.30	0.006	-0.31	0.006
TG/TC	0.35	0.001	-0.35	0.001
AIP	0.36	0.001	-0.36	0.006
Adiponectin/TNF α ratio	-0.45	0.001	0.45	0.001

3

OPG

12

GDM OPG
Akinci GDM

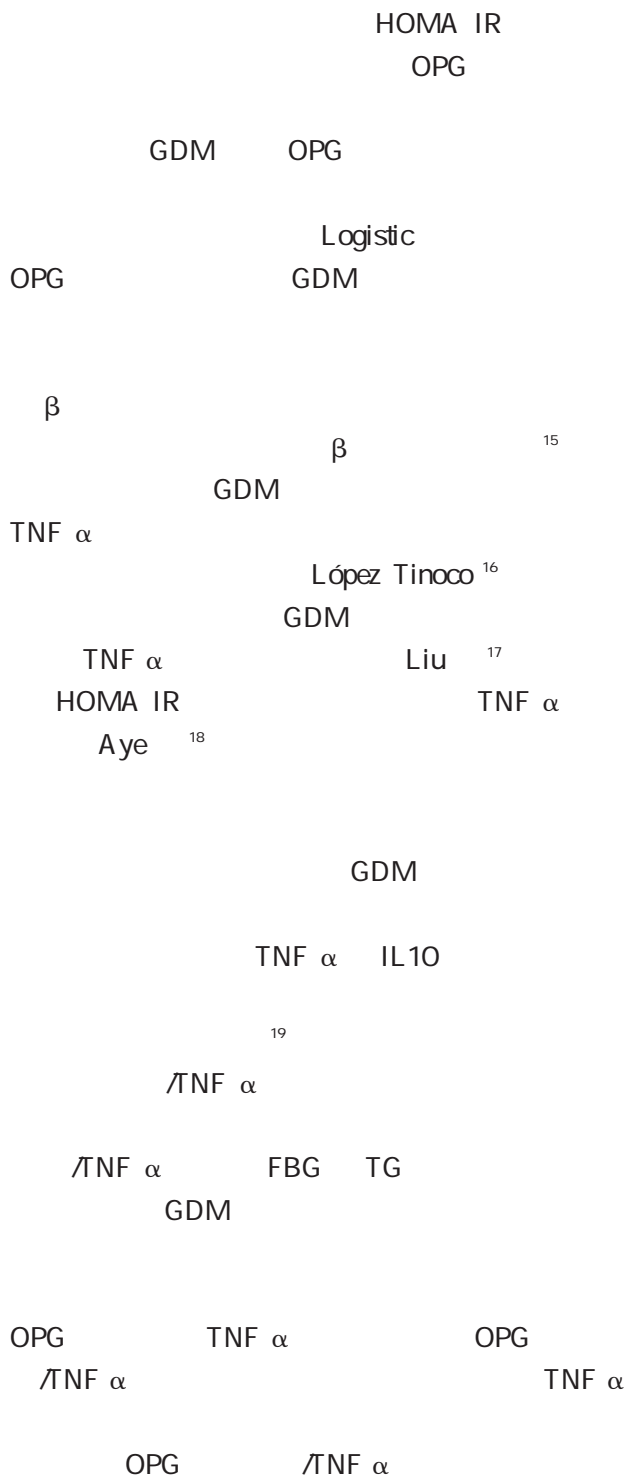
OPG BMI
HOMA IR C
OPG
1 TNF α
OPG
GDM

13

14

OPG

OPG TNF α $\bar{x} \pm s$



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• •

CRP sCD14 ST

*

CD14	sCD14 ST			2016	12	C	2019	3	CRP
	164				CRP	sCD14 ST			logistic
sCD14 ST						ROC			CRP
						22			13.41
									P<0.05
T2 T3 T4		CRP	sCD14 ST						
		CRP	sCD14 ST						
P<0.05	logistic								CRP
sCD14 ST				P<0.05	ROC		CRP	sCD14 ST	
	AUC	0.712	0.804	0.918	71.02	79.55	87.22	72.27	77.48
85.96									
CRP sCD14 ST									
				C			CD14		

Nosocomial infection factors and prediction of CRP and sCD14-ST in patients with rheumatoid arthritis

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 463000

ABSTRACT Objective To study the risk factors of rheumatoid arthritis complicated with nosocomial infection. Hospital 1 to 1000 patients with nosocomial infection were included. Control group

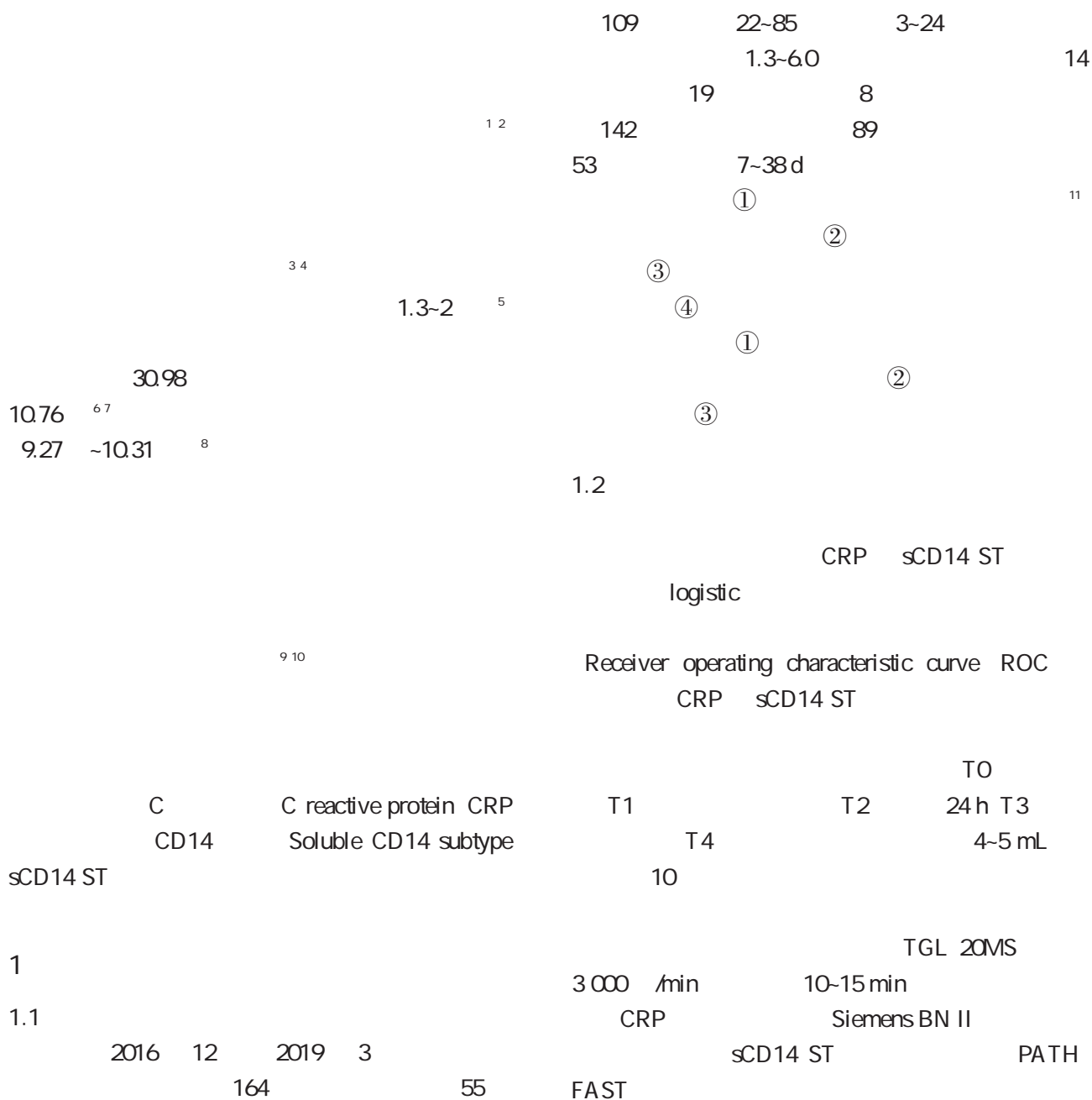
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creased first and then decreased while those without nosocomial infections continued to decline. The levels of CRP and sCD14 ST in patients with nosocomial infections were higher than those without nosocomial infections $P < 0.05$. Multivariate logistic regression analysis confirmed that diabetes mellitus invasive operation catheterization immunosuppressive agents hospitalization time and plasma CRP and sCD14 ST were risk factors for nosocomial infection in rheumatoid arthritis $P < 0.05$. ROC curve showed that CRP sCD14 ST and their combined detection AUC were 0.712 0.804 0.918 respectively sensitivity were 71.02% 79.55% 87.22% respectively specificity were 72.27% 77.48% 85.96% respectively. Conclusion There are various risk factors for rheumatoid arthritis complicated with nosocomial infection. Strengthening screening of plasma CRP and sCD14 ST levels are helpful for early detection early intervention and reduction of nosocomial infection rate.

KEY WORDS Rheumatoid arthritis Nosocomial infection Risk factors C reactive protein Soluble CD14 subtype Predictive value



2.3

1.3 SPSS 24.0 TO T1 CRP sCD14 ST
 $\bar{x} \pm s$ χ^2 LSD t n CRP sCD14 ST
 logistic ROC CRP sCD14 ST T2 T3 T4 CRP
 Area under the AUC<0.50 P<0.05 2
 AUC=1.0
 P<0.05
 2
 2.1 22 13.41 22
 12 54.55%
 7 31.82% 2 9.09%
 1 4.55%
 2.2
 P<0.05 1

2.4

\$ logistic
 " ž " ' & O T

		AUC			
CRP	25.10 mg/L	0.712	71.02	72.27	
sCD14 ST	801.80 pg/mL	0.804	79.55	77.48	
CRO	sCD14 ST	-	0.918	87.22	85.96

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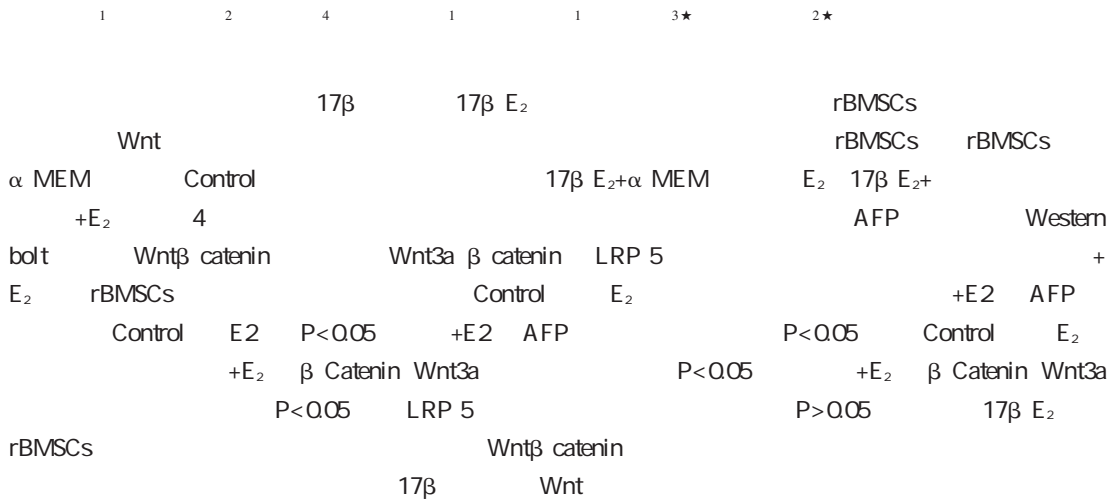
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17β

rBMSCs

Wnt



Regulation of 17β -estradiol on the differentiation of hepatic progenitor cells and Wnt signaling pathway in rBMSCs

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ABSTRACT Objective To investigate the regulatory effect of 17β estradiol 17β E2 on the differ entiation of hepatic progeni tor cells of rat bone marrow mesenchymal stem cells rBMSCs and Wnt signaling pathway. Methods rBMSCs were obtained by whole bone marrow adherence method. The cells were divided into four groups α MEM medium control liver inducing medium classical and E2+α. MEM medium E2 E2 + hepatic inducing medium classical + E2 observed in morphology. The AFP concentration was measured using the luminescence immunoassay. The expression of Wnt signaling proteins included Wnt3a β

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catenin and LRP 5 was detected by Western blot. Results Morphologically rBMSCs in the classic and classic + E2 groups gradually showed liver progenitor like changes while the control group and E2 group showed no significant changes. The AFP concentration of classic group and classic + E2 group was significantly higher than that of the control group and E2 group P<0.05 . The AFP value of classic + E2 group was significantly higher than that of the classic group P<0.05 . Compared with the control group and the E2 group the expression levels of β Catenin and Wnt3a in the classic group and the classic + E2 group were significantly reduced P<0.05 and the expression levels of β Catenin and Wnt3a in the classic + E2 group were significantly lower than that in the classic group P<0.05 . There was no statistically significant difference in the expression of LRP 5 P>0.05 . Conclusion 17 β E2 can promote the differentiation of rBMSCs hepatic progenitor cells and the mechanism may be related to the regulation of Wnt β catenin signaling pathway.

KEY WORDS Bone mesenchymal stem cells 17 β estradiol Wnt signaling pathway Differentiation of hepatic progenitor cells

artificial liver support system ALSS

CD45 legend β Catenin GAPDH CST CO₂

CD80 Tris HCL pH 8.3 Wnt3a WB Thermo Scientific3111

CD90 LRP 5 SW CJ IF

Bio Legend

1.2

Bone marrow mesenchymal stem cells BMSCs

Bio Rad680 SAria ScanDrop 100

BD FAC Boyue 17 4070

Tanon 1600 HE 120

3.6

BMSCs

1.3

1.3.1

row mesenchymal stem cells rBMSCs

P₃ P₅ rBMSCs 25 cm²

rBMSCs 37 5% CO₂

17 β 17 β estradiol 3 d 1 2

17 β E2 rBMSCs 4 α MEM

17 β E₂ Wnt Control 15% FBS

rBMSCs 10⁻⁶ mol/L 17 β E₂+ α MEM

1 E₂ 10⁻⁶ mol/L 17 β E₂+
+E₂

1.1 P₃ P₅ rBMSCs D Hanks

rBMSCs 2 9%

1.2 2 000 6 6

α MEM HyClone 10 200 L μ 37

D Hanks 5% CO₂ 1 6

PanEra CCK 8 100 L μ

0.25% Gibco 17 β 10 L CCK μ 8 25 h

CCK 8 Dojindo CD29 450 nm

OD OD

rBMSCs 1A rBMSCs S

1.3.2 2d 8d 1B rBMSCs

2000 rpm 6 min

D Hanks 1B 1d 1A d 300 1d E Ep @ GMP 1A S ñ 0 2€ 44 P

1.5 mL 700 L µ

4 D Hanks

2 500 L µ 1 600 rpm 10

min 200 L PI µ

15 min

1.3.3 rBMSCs

1 × 10⁶ cells/

mL CD29 CD45 CD80

CD90 37 30 min

1.3.4 rBMSCs

1.4.1 60%

D Hanks

3 95% 10 min

3 0.1% Tris HCL

O 37 45 min 60 min

Tris HCL 60%

O

1.3.5 rBMSCs AFP

1.4.1 1.4.2 3 6 10

4 300 L µ

AFP

1.3.6 Wnt

1.4.1 1.4.2

1 4

99 10 min

β Catenin Wnt3a

LRP 5 4 37 2h

1.3.7

SPSS 13.0

$\bar{x} \pm s$

Bon

ferroni P<0.05

2

2.1 rBMSCs

rBMSCs

2.5 rBMSCs

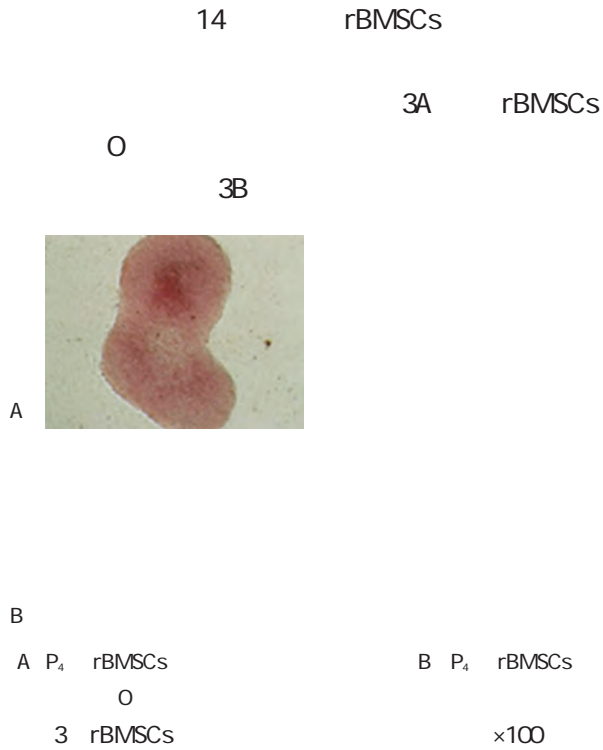


Figure 3 Identification of differentiation potency of rBMSCs from P₄ generation ×100

2.7 rBMSCs

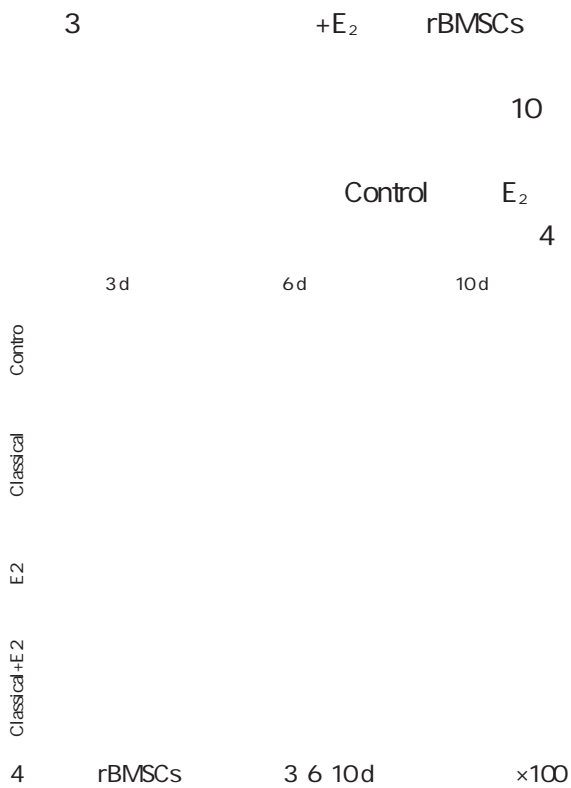


Figure 4 Cell morphology after 3 6 and 10 days of rBMSCs induction in each group ×100

2.8 rBMSCsAFP

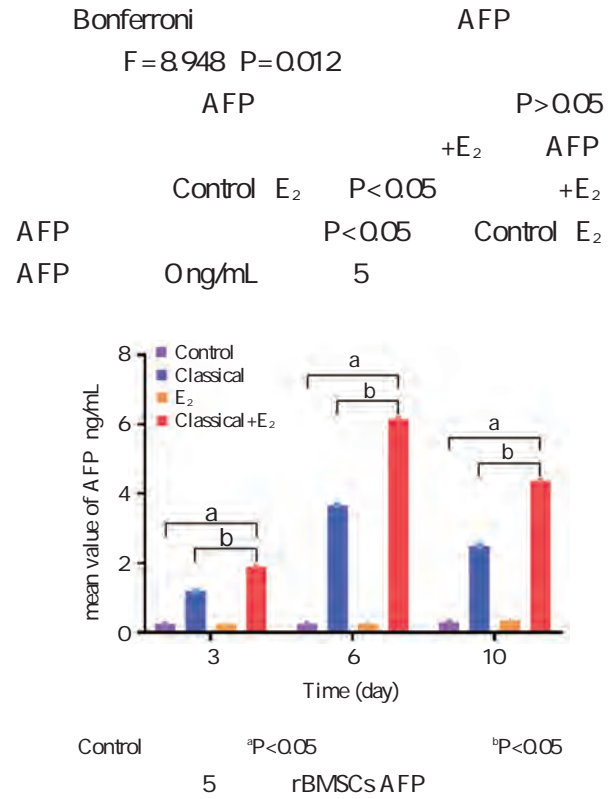
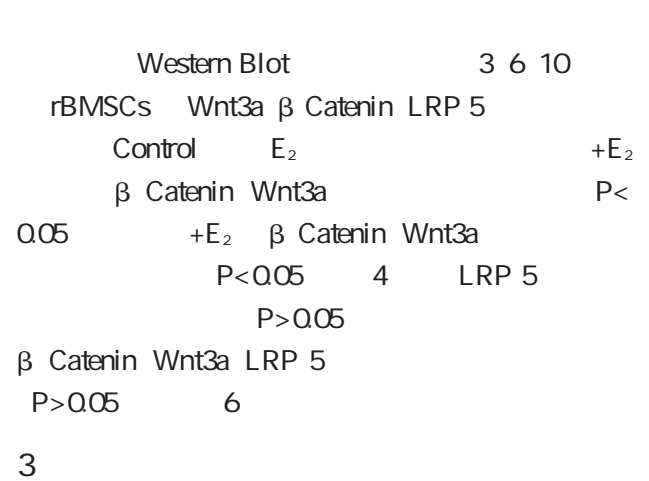


Figure 5 Average AFP of rBMSCs in each group

2.9 rBMSCs



BMSCs

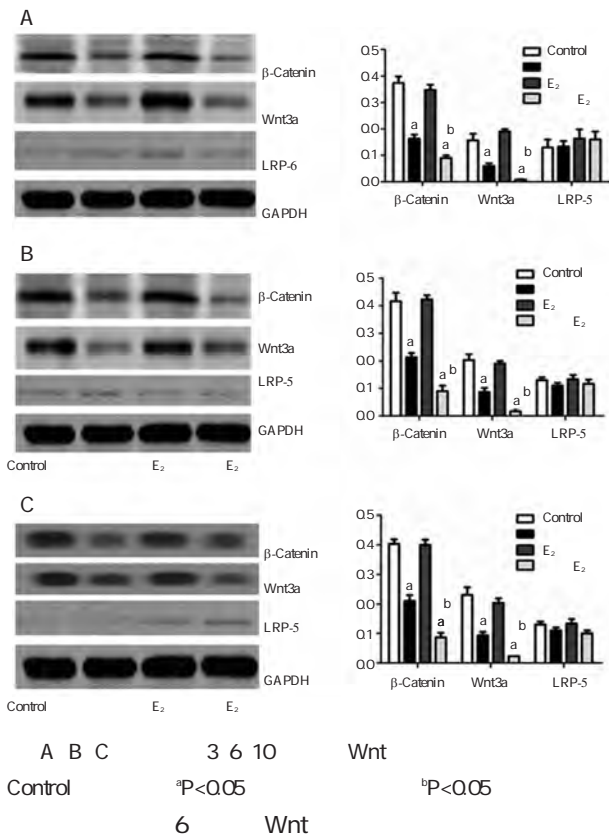
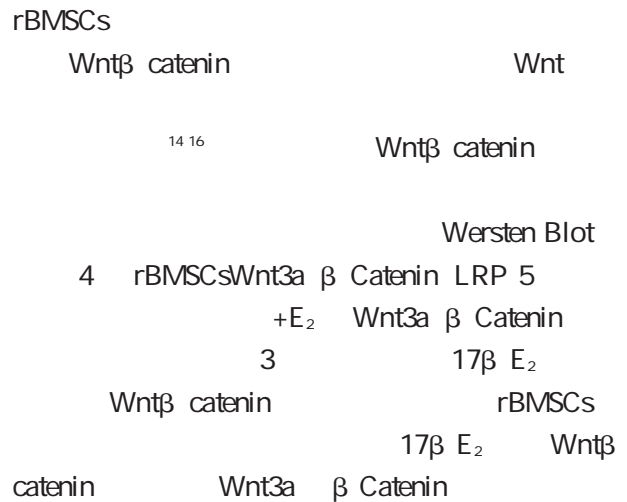
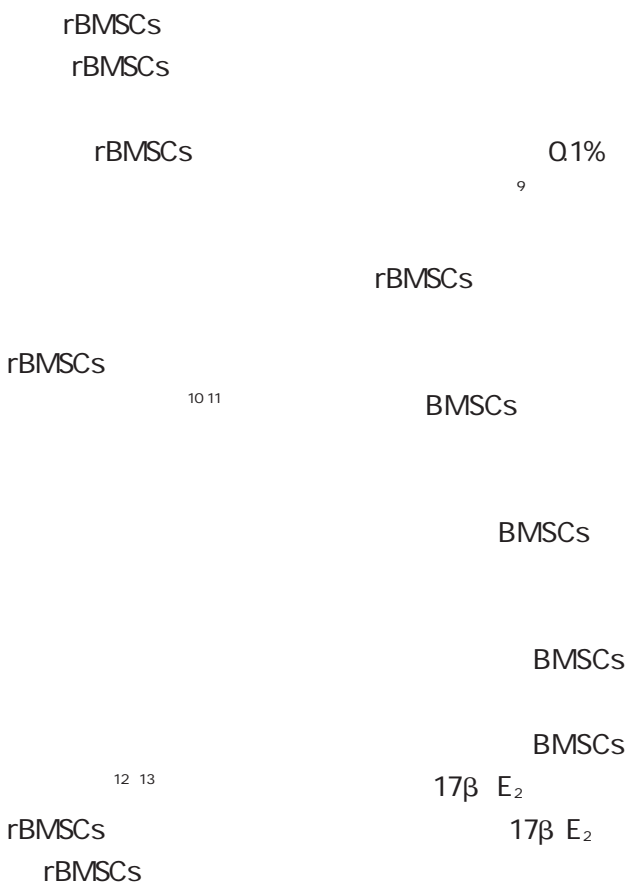
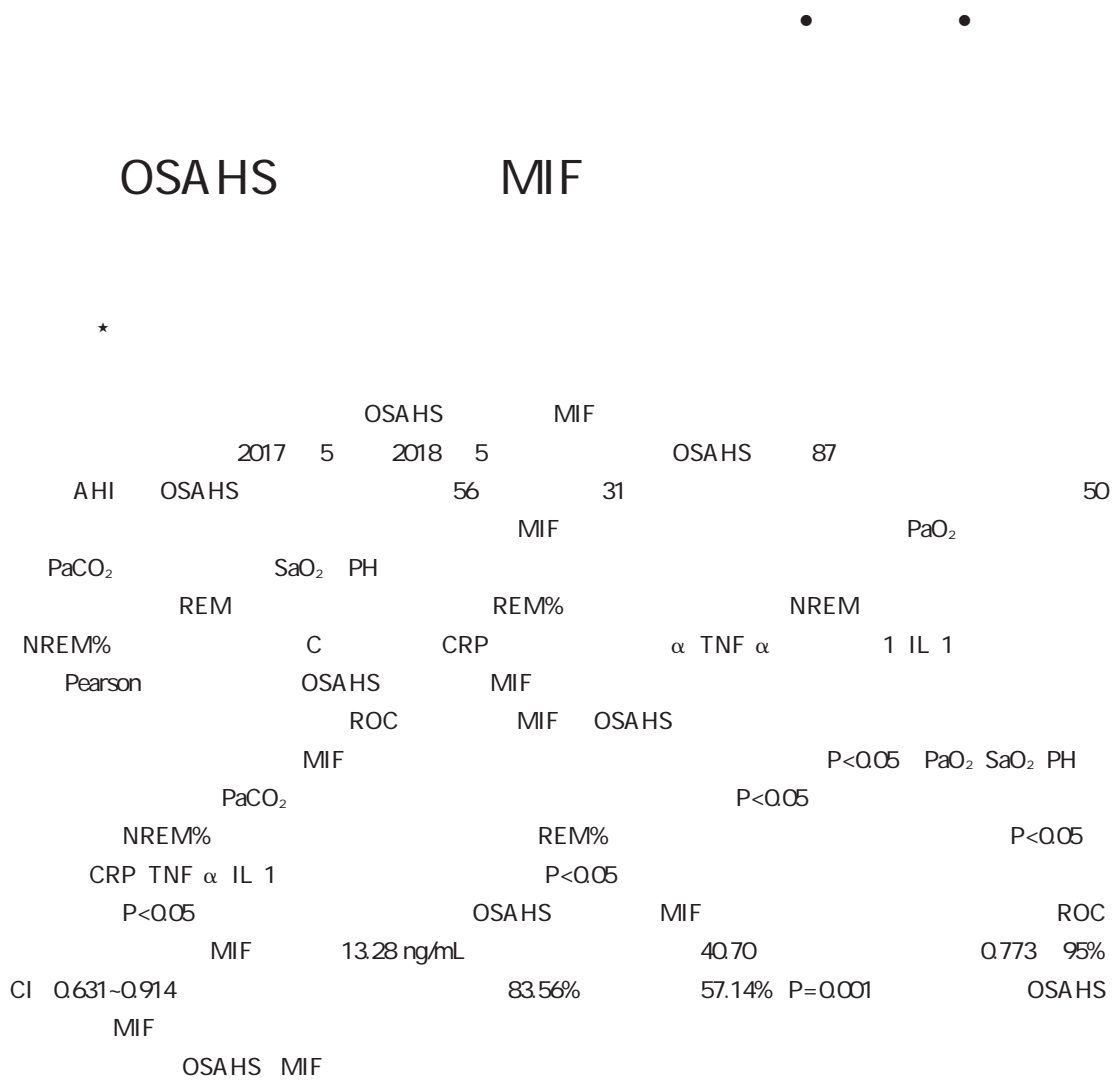


Figure 6 Expression of Wnt signaling protein in each group



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Estimated value of serum MIF level in assessing hypoxia sleep disorder and vascular endothelial injury in adult OSAHS patients

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ABSTRACT Objective To investigate the correlation between serum macrophage migration inhibiting factor (MIF) level and severity of in adult OSAHS patients and its predictive value to long term risk of cerebral infarction in patients. Methods 87 cases of adult OSAHS patients in our hospital during May 2017 to May 2018 were divided into mild to moderate group (n=56) severe group (n=31) according to apnea hypopnea index (AHI). The differences in serum MIF, arterial blood gas indicators, Blood oxygen partial pressure, and h Wd W. Results The MIF level in the severe group was significantly higher than that in the mild to moderate group (P<0.001). The differences in arterial blood gas indicators, Blood oxygen partial pressure, and h Wd W were not significant (P>0.05). Conclusion The MIF level in adult OSAHS patients is significantly correlated with the severity of hypoxia sleep disorder and vascular endothelial injury. MIF level can be used as a reference for the assessment of hypoxia sleep disorder and vascular endothelial injury in adult OSAHS patients.

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cytokines C reactive protein CRP tumor necrosis factor alpha TNF alpha Interleukin 1 IL 1 were compared among the subjects in each group. Pearson test was used to evaluate the correlation between serum MIF level and hypoxia sleep disorder and vascular endothelial injury in adult OSAHS patients. The ROC curve was used to analyze the predictive value of MIF for long term cerebral infarction in OSAHS patients. Results Serum MIF level of mild to moderate group severe group were higher than those in normal control group PaO₂ SaO₂ and PH levels were lower than normal control group PaCO₂ level was higher than that in normal control group Sleep latency total time of awakening number of awakening and NREM% were greater than those in normal control group while total sleep time and REM% were smaller than those in normal control group serum levels of CRP TNF α IL 1 were higher than those in normal control group level of NO was lower than that of normal control group P<0.05 . Changes of above indexes in severe group were all greater than those in mild and moderate group P<0.05 . Correlation analysis showed that serum MIF level of adult OSAHS patients was directly correlated with the severity of disease. ROC curve showed that when serum MIF level was 13.28 ng/mL the maximum yoden index was 40.70 and the area under the curve was 0.773 95% CI 0.631-0.914 . The sensitivity and specificity of predicting the occurrence of cerebral infarction were 83.56% and 57.14% P=0.001. Conclusion Serum MIF level of adult OSAHS patients increases abnormally which has certain value in reflecting the severity of disease.

KEY WORDS OSAHS MIF Arterial blood gases Sleep structure Endothelial injury Inflammatory response Cerebral infarction

Obstruc 2011 4 OSAHS ②
 tive sleep apnea hypopnea syndrome OSAHS 18-79 ③ OSAHS
 ①
 ②
 1 2 ③
 OSAHS ④
 Macrophage migration inhibitor MIF
 3 Apnea hypopnea Index AHI OSAHS
 5 h≤AHI<30 h 56
 AHI≥30 31
 1.2 MIF
 3 8 00am
 OSAHS OSAHS
 MIF 3.0mL 37 2h
 OSAHS HBS 1101 BioRad
 1 HumanMIF ELISA Kit MIF
 1.3
 3 8 00am
 2017 5 2018 5 2.0mL GEM3000
 OSAHS 87 Oxygen partial pressure PaO₂
 AHI OSAHS 56 Partial pressure of carbon dioxide Pa
 31 50 CO₂ Oxygen saturation SaO₂ PH
 21~59 44.76±
 7.65 28 22 ① Alice4

Rapid eye movemenent

REM REM% $\bar{x} \pm s$ LSD t n
 Non Rapid Eye Movement NREM
 NREM% % χ^2
 HBS 1101 BioRad Pearson MIF
 C Receiver operating char
 C reactive protein CRP Human CRP ELISA acteristic curve ROC P<0.05
 Kit α Tumor necrosis fac
 tor α TNF α Human TNF α ELISA Kit 2
 1 Interleukin 1 IL 1 Human IL 1 2.1 MIF
 ELISA Kit 1.4 MIF
 MIF
 P<0.05 PaO₂
 2019 10 31 SaO₂ PH PaCO₂
 1.5 SPSS 20.0 P<0.05 1

Table 1 Comparison of serum MIF and arterial blood gas index levels between the 3 groups $\bar{x} \pm s$

	n=50	n=56	n=31	F/ χ^2	P
/	28/22	34/22	22/9	1.822	0.402
	44.76±7.65	44.83±8.29	45.10±8.32	0.652	0.516
	24.50±3.74	24.87±3.49	24.76±3.52	0.782	0.436
MIF	8.59±0.97	11.51±0.76	13.73±0.98	16.454	0.000
PaO ₂	98.63±1.23	92.20±5.81	83.64±9.52	7.725	0.000
PaCO ₂	38.46±5.10	44.80±6.37	50.72±7.34	9.470	0.000
SaO ₂	98.77±1.20	94.65±3.42	85.84±9.20	10.711	0.000
PH	7.39±0.11	7.28±0.18	7.13±0.24	8.295	0.000

2.2 REM% P<0.05
 NREM% 2

Table 2 Comparison of sleep structure parameters between 3 groups $\bar{x} \pm s$

	n	min	min	min	REM%	NREM%	
	50	30.15±4.28	439.74±73.13	57.12±8.59	5.46±0.68	22.38±2.75	73.10±8.65
	56	43.84±5.61	380.31±45.82	79.66±9.21	8.20±0.92	19.45±2.17	82.64±9.53
	31	57.96±8.22	311.49±37.24	93.74±11.85	9.76±1.14	14.02±1.83	87.92±9.70
F		15.914	10.920	15.549	12.845	15.372	8.055
P		0.000	0.000	0.000	0.000	0.000	0.000

2.3

3 3 $\bar{x} \pm s$
 Table 3 Comparison of serum inflammatory cytokine levels
 between 3 groups $\bar{x} \pm s$

	n	CRP mg/L	TNF α (pg/mL)	IL 1 (pg/mL)
	50	1.05±0.23	3.46±0.48	2.81±0.35
	56	4.28±0.59	9.21±1.52	5.03±0.57
	31	10.64±2.12	17.58±2.63	9.74±1.13
F		29.022	35.634	34.385
P		0.000	0.000	0.000

2.4

OSAHS MIF PaO₂ SaO₂ PH
 PaCO₂
 NREM%
 REM% CRP TNF α 3
 IL 1 P<0.05 4 OSAHS

2.6 ROC

87 OSAHS
 14 MIF 13.28 ng/mL
 40.70 0.773
 95%CI 0.631-0.914
 83.56% 57.14% P=0.001
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PCT NT proBNP

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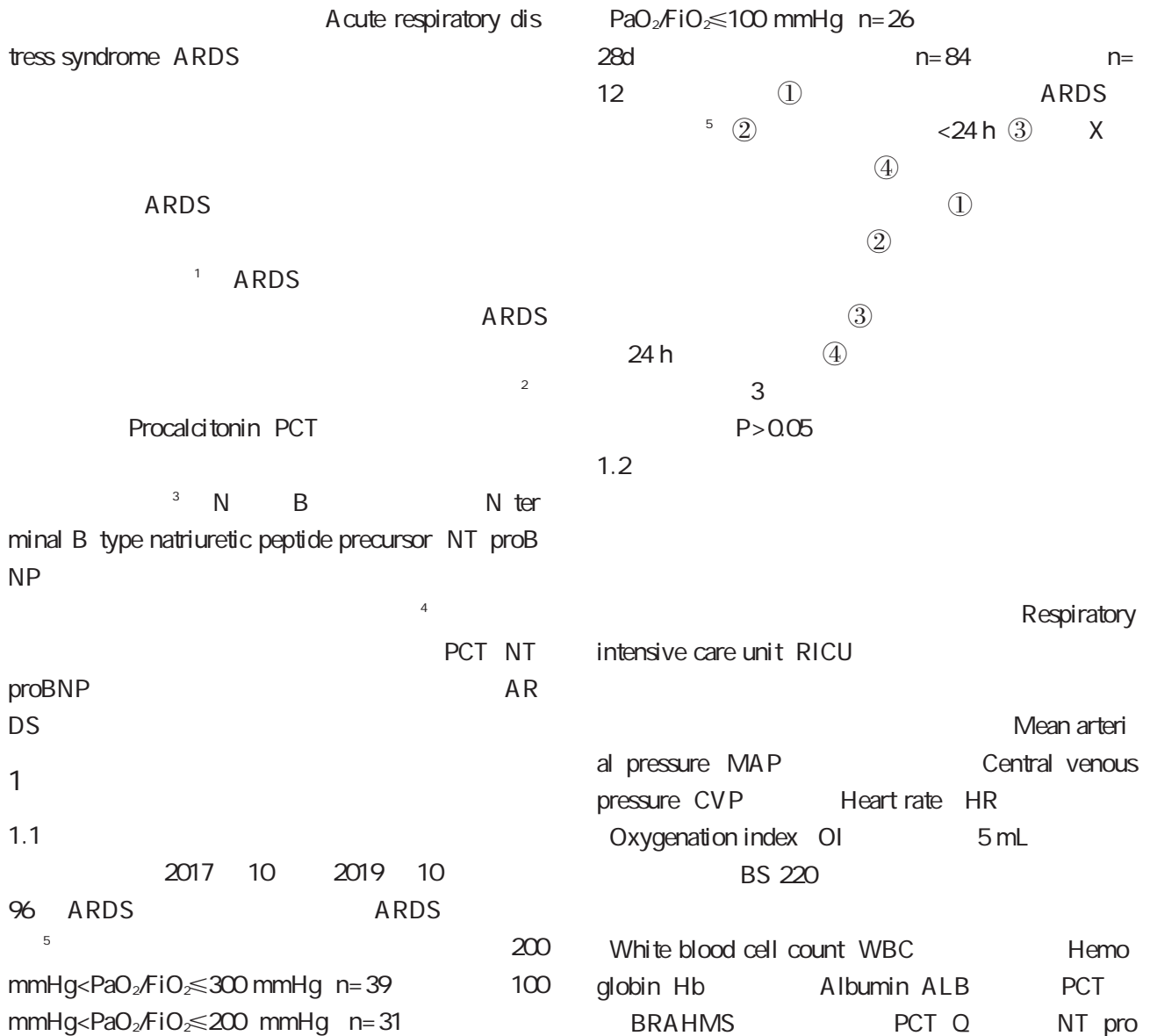
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and NT pro BNP in the prognosis diagnosis of ARDS patients. Results Differences in gender age respiratory rate oxygen concentration mean arterial pressure MAP central venous pressure CVP white blood cell count WBC hemoglobin Hb albumin ALB in mild moderate and severe groups no statistical significance $P>0.05$. There were statistically significant differences in hospitalization time mechanical ventilation time oxygenation index OI heart rate HR PCT NT pro BNP SOFA and APACHE II scores in the mild moderate and severe groups $P<0.05$. There was a significant positive correlation between PCT and SOFA and APACHE II scores and a significant negative correlation between NT pro BNP and SOFA and APACHE II scores $P<0.05$. The differences in serum PCT and NT pro BNP levels between the survival group and the death group were statistically significant $P<0.05$. ROC curve results showed that the AUC of PCT NT pro BNP and the combined diagnosis were 0.847 95% CI 0.795-0.900 0.729 95% CI 0.657-0.802 0.932 95% CI 0.899-0.965. Conclusion Serum PCT and NT pro BNP have significant value in predicting the severity and prognosis of patients with ARDS and can be used as reference indicators for early diagnosis of ARDS.

KEY WORDS Acute respiratory distress syndrome Procalcitonin N terminal B type natriuretic peptide precursor



BNP		ELISA		P<0.05		RICU	
28d		Sequential Or		1		1	
gan Failure Assessment SOFA		II acute physiology and chronic health evaluation scoring system APACHE II		Table 1 Comparison of baseline data of patients with ARDS of different severity $\bar{x} \pm s$		$\bar{x} \pm s$	
1.3		SPSS 19.0		n		RICU	
n		$\bar{x} \pm s$		t		d	
APACHE II		PCT NT proBNP		Spearman		d	
ROC		AUC		PCT NT		d	
pro BNP ARDS		P<0.05		2.2		ARDS	
0.05		MAP CVP WBC Hb ALB		P>0.05		OI	
2		HR PCT NT pro BNP SOFA APACHE II		P<0.05		P<0.05	
2.1		ARDS		OI		HR PCT NT pro BNP	
		P>0.05 3		RICU		SOFA APACHE II 2	
		2		ARDS		$\bar{x} \pm s$	
		Table 2 Comparison of clinical data of patients with ARDS of different severity $\bar{x} \pm s$					
		n=39		n=31		n=26	
		F		P			
/min		29.8±4.0		31.5±4.4		31.6±3.8	
L/min		0.6±0.2		0.7±0.4		0.7±0.5	
OI		118.2±9.9		96.4±7.8		84.3±8.3	
MAP mmHg		85.3±11.5		84.4±10.9		81.8±11.7	
CVP cmH ₂ O		9.3±2.9		8.7±3.1		8.3±3.6	
HR /min		93.8±7.2		98.3±8.1 ^a		113.1±11.4 ^{ab}	
WBC ×10 ⁹ /L		6.7±1.8		7.2±2.0		7.5±2.6	
Hb g/L		116.8±22.4		113.8±21.9		109.8±24.5	
ALB g/L		28.8±5.3		27.6±4.6		27.0±5.2	
PCT ng/mL		5.2±1.7		7.2±1.8 ^a		9.6±2.3 ^b	
NT pro BNP ng/L		1399.5±164.2		2427.8±207.5 ^a		4438.6±369.7 ^{ab}	
SOFA		7.1±1.7		8.7±1.1 ^a		10.1±3.2 ^{ab}	
APACHE II		15.3±3.6		19.2±4.1 ^a		25.2±3.9 ^b	
		^a P<0.05		^b P<0.05			
2.3 ARDS		PCT NT pro BNP		0.485 P=0.000			
APACHE II SOFA		PCT SOFA r=0.492 P=0.000		APACHE II		P<0.05	
r=0.473 P=0.000		NT pro		2.4		ARDS	
BNP SOFA r=0.416 P=0.000		APACHE II r=		PCT		PCT NT pro BNP	

NT pro BNP

P<0.05 3

3 ARDS PCT NT pro BNP

$\bar{x} \pm s$

Table 3 Comparison of serum PCT and NT-pro BNP levels in patients with ARDS with different prognosis $\bar{x} \pm s$

	n	PCT (ng/mL)	NT pro BNP (ng/L)
	84	6.6±1.9	1576.3±163.5
	12	10.3±2.8	4722.1±405.2
t		4.434	26.586
P		0.001	0.000

2.5 PCT NT pro BNP ARDS

ROC

ROC PCT NT pro BNP

AUC 0.847 95% CI 0.795-0.900

0.729 95% CI 0.657-0.802 0.932 95% CI 0.899-0.965

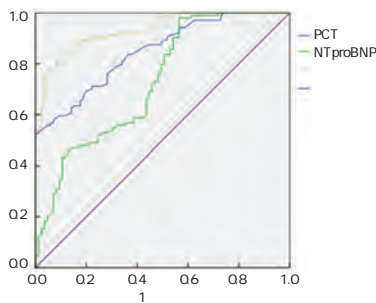
PCT 6.3 ng/mL

0.835 0.759 NT pro BNP

2794.5 ng/L 0.721

0.729 0.913

0.906 1



1 PCT NT pro BNP ARDS

ROC

Figure 1 ROC curve of PCT and NT pro BNP for prognosis diagnosis of ARDS patients

3

ARDS

ARDS

ARDS 8

ALI 67

ARDS

ARDS

ARDS

AR

DS

HR MAP CVP

ARDS

9

PCT 116

C PCT

PCT PCT PCT

10 11

PCT PCT

BNP

NT pro BNP NT pro BNP BNP

12 NT pro BNP ARDS

13 NT pro BNP

ARDS 14

BNP

15 IL 6 TNF α

BNP

NT pro BNP

PCT NT pro BNP ARDS

ARDS

1 Hendrickson CM Matthay MA. Potential Value of Biomarker Signatures in Sepsis and Acute Respiratory Distress Syn

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4 HE4 9 CXCL9 LN 80
2016 1 ~2018 1 82 LN
SLE LN HE4 CXCL9 6
ROC HE4 CXCL9
1
6 HE4 CXCL9 Logistic LN ROC
HE4 CXCL9 HE4 CXCL9
P<0.05 4 9HE4CXCL

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CL9 in the study group were higher than those in the control group $P < 0.05$. The ROC curve analysis showed that the combined AUC of serum HE4 and CXCL9 was 0.773 which was greater than HE4 0.670 and CXCL9 0.613 when the cutoff value of HE4 > 160.07 U/mL and the cutoff value of CXCL9 > 218.93 pg/mL the sensitivity was 76.83% and the specificity was 76.25% the serum HE4 and CXCL9 levels were higher than those with good prognosis after 6 months of treatment $P < 0.05$. Logistic regression analysis showed that SLE DAI score OR=2.820 serum HE4 OR=1.283 and CXCL9 OR=2.141 levels were important factors affecting the prognosis of LN $P < 0.05$ the ROC curve analysis showed that CXCL9 predicted a poor prognosis AUC of 0.670 after treatment for 6 months which was greater than HE4 0.667 when the cutoff value was > 191.88 the sensitivity was 60.00% and the specificity was 76.12%. Conclusion Serum HE4 and CXCL9 are highly expressed in LN. Combined detection has good diagnostic value and is an important prognostic factor. It can be used as a predictor of poor prognosis.

KEY WORDS Lupus nephritis Chemokine ligand 9 Human epididymis protein 4 Composite renal endpoint

Lupus nephritis LN
Systemic lupus erythematosus SLE

12 LN

3 SLE

9

4 SLE

4 Human epi

5 LN

HE4 CXCL9 LN

1

1.1

LN	2016	1	~2018	1	82
20-63			12		70
			35.31±7.33		
	126.30±35.27		mol/L μ		9.22±
2.05 mmol/L		80			
SLE	LN				11
69	21-64		36.48		

eGFR $\geq 50\%$ >218.93 pg/mL 76.83%
 1.4 76.25% 2
 2.3
 SPSS 24.0 1
 $\bar{x} \pm s$ t n % χ^2 15 2 8
 Logistic LN eGFR $\geq 50\%$
 ROC HE4 CXCL9 LN
 P<0.05 P>0.05 SLE
 SLEDAI P<0.05
 2 3

2.1 HE4 CXCL9
 HE4 CXCL9
 P<0.05 1
 1 HE4 CXCL9 $\bar{x} \pm s$
 Table 1 Comparison of serum HE4 and CXCL9 levels in two groups at admission $\bar{x} \pm s$

n	HE4 U/mL	CXCL9 pg/mL
82	184.35±62.24	234.46±42.09
80	126.27±58.82	207.11±38.20
t	6.101	4.328
P	<0.001	<0.001

3 $\bar{x} \pm s$
 Table 3 Comparison of General information of patients with different prognosis $\bar{x} \pm s$

	n=67	n=15	t/ χ^2	P
	35.14±7.06	36.09±6.57	0.477	0.635
	1.60±0.49	1.74±0.61	0.955	0.342
/	10/57	2/13	0.061	0.805
SLEDAI	12.80±2.33	17.16±2.51	6.461	0.001

2.2 HE4 CXCL9 HE4 CXCL9
 ROC HE4 CXCL9 P>0.05 6
 AUC 0.773 HE4 0.670 CXCL9 HE4 CXCL9 P<0.05
 0.613 HE4 >160.07 U/mL CXCL9 4

2 ROC
 Table 2 ROC analysis results

	AUC	95%CI	Z	P		%	%
HE4	0.670	0.588-0.753	4.044	<0.001	>162.08 U/mL	45.12	81.25
CXCL9	0.613	0.526-0.700	2.559	0.011	>221.07 pg/mL	75.61	43.75
	0.773	0.697-0.848	7.092	<0.001	HE4>160.07 U/mL CXCL9>218.93 pg/mL	76.83	76.25

4 6 HE4 CXCL9 $\bar{x} \pm s$
 Table 4 Serum HE4 and CXCL9 levels at admission to patients with different prognosis at 6 months after treatment $\bar{x} \pm s$

	n	HE4 U/mL		CXCL9 pg/mL	
		4	6	6	6
	15	193.39±35.49	137.58±27.25	247.73±38.84	201.85±33.51
	67	182.32±31.26	116.19±24.17	231.49±34.77	174.31±28.26
t		1.210	3.027	1.601	3.297
P		0.230	0.05	0.113	0.05

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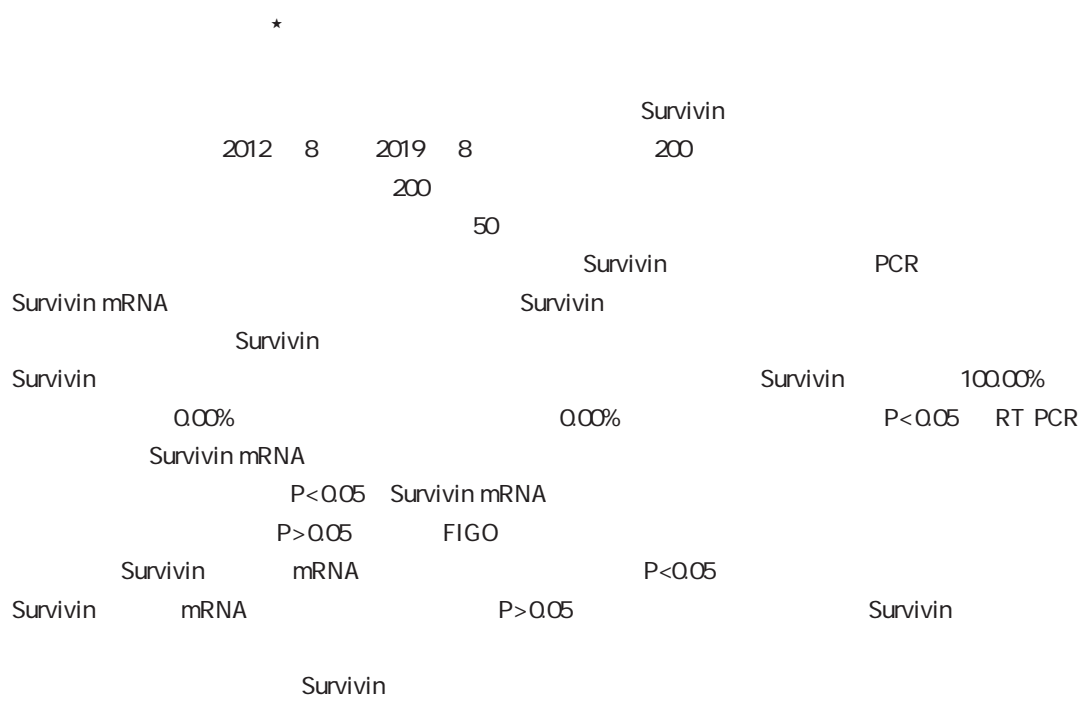
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14 J . 2019 35 11 1813 1815.

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Survivin



The expression of Survivin in cervical phosphorous carcinoma and its relationship with clinicopathological features

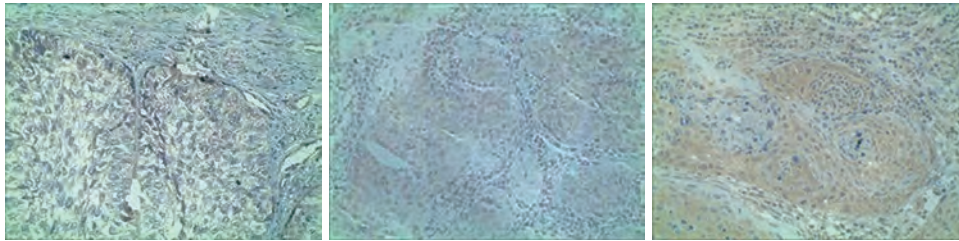
Eerdemutu JIA Yuling* QIN Xiaoling NIU Li rong BAO Ying na YU Zhilong
 Department of radiotherapy affiliated hospital of Inner Mongolia medical university Hohhot Mongolia
 China 010050

ABSTRACT Objective To investigate the expression of Survivin and its relationship with clinicopathological features of cervical phosphorous carcinoma. Methods From August 2012 to August 2019 200 cases of cancer tissue samples from cervical phosphorus cancer patients were collected and 200 cases of paracancerous tissues confirmed by pathological examination as normal cervical tissues were taken as controls. In addition 50 cases of cervical squamous intraepithelial lesions confirmed by pathology after cervical tumor resection were selected. Immunohistochemical staining was used to detect the expression of survivin protein in cervical squamous cell carcinoma tissues paracancerous tissues and cervical squamous intraepithelial lesion. The expression of survivin mRNA in each tissue was detected by PCR. The relationship between the expression of Survivin and the clinicopathological features of cervical phosphorous carcinoma was

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1.4 RT-PCR Survivin 2
 mRNA TRIzol 2.1 Survivin
 RNA cDNA PCR Survivin
 GAPDH 2^{Ct} ΔΔ 0.00% Survivin
 1.5 SPSS 20.00 n Survivin
 $\bar{x} \pm s$ t Survivin
 P < 0.05 P < 0.05
 1 z " ' "



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LP a OPN IRF 4

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 115 4 IRF 4 DHVD LP a OPN IRF 4 mRNA LVEF CI
 120 DHVD LP a OPN IRF 4 mRNA LVEF CI
 F=47.427 66.514 36.432 P<0.05
 OPN IRF 4 mRNA DHVD LP a DHVD LP a
 AUC 0.802 70.00% 86.09% 3 LP
 a OPN IRF 4 mRNA LVEF CI P<0.05
 LP a OPN LVEF CI IRF 4 mRNA LVEF CI
 P<0.05 DHVD LP a OPN IRF 4 mRNA DHVD
 DHVD
 a 4

Expressions and clinical significance of serum LP a OPN IRF-4 in patients with degenerative heart valve disease

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Department of cardiovascular surgery The First affiliated hospital of xinxiang medical college Weihui Henan China 453100

ABSTRACT Objective To investigate the expression of serum lipoprotein a LP a osteopontin OPN interferon regulatory factor 4 IRF 4 in patients with degenerative heart valve disease DHVD and its clinical significance. Methods 120 patients with DHVD in our hospital were selected as the study group and 115 healthy people who were examined during the same period were used as the control group. The serum LP a OPN IRF 4 mRNA levels were compared between the two groups. The influencing factors of the incidence of DHVD and the diagnostic value of serum indicators for DHVD were analyzed. The levels of serum LP a OPN IRF 4 mRNA and cardiac function indexes left ventricular ejection fraction LVEF cardiac index CI of patients with different therapeutic effects after 3 months of treatment were compared. The correlation between serum indexes and cardiac function indexes was evaluated. Results The serum LP a OPN IRF 4 mRNA levels have significant difference in the patients with multi valvular disease and with single valvular disease and healthy people F=47.427 66.514 36.432 P<0.05 . Coronary heart disease smoking

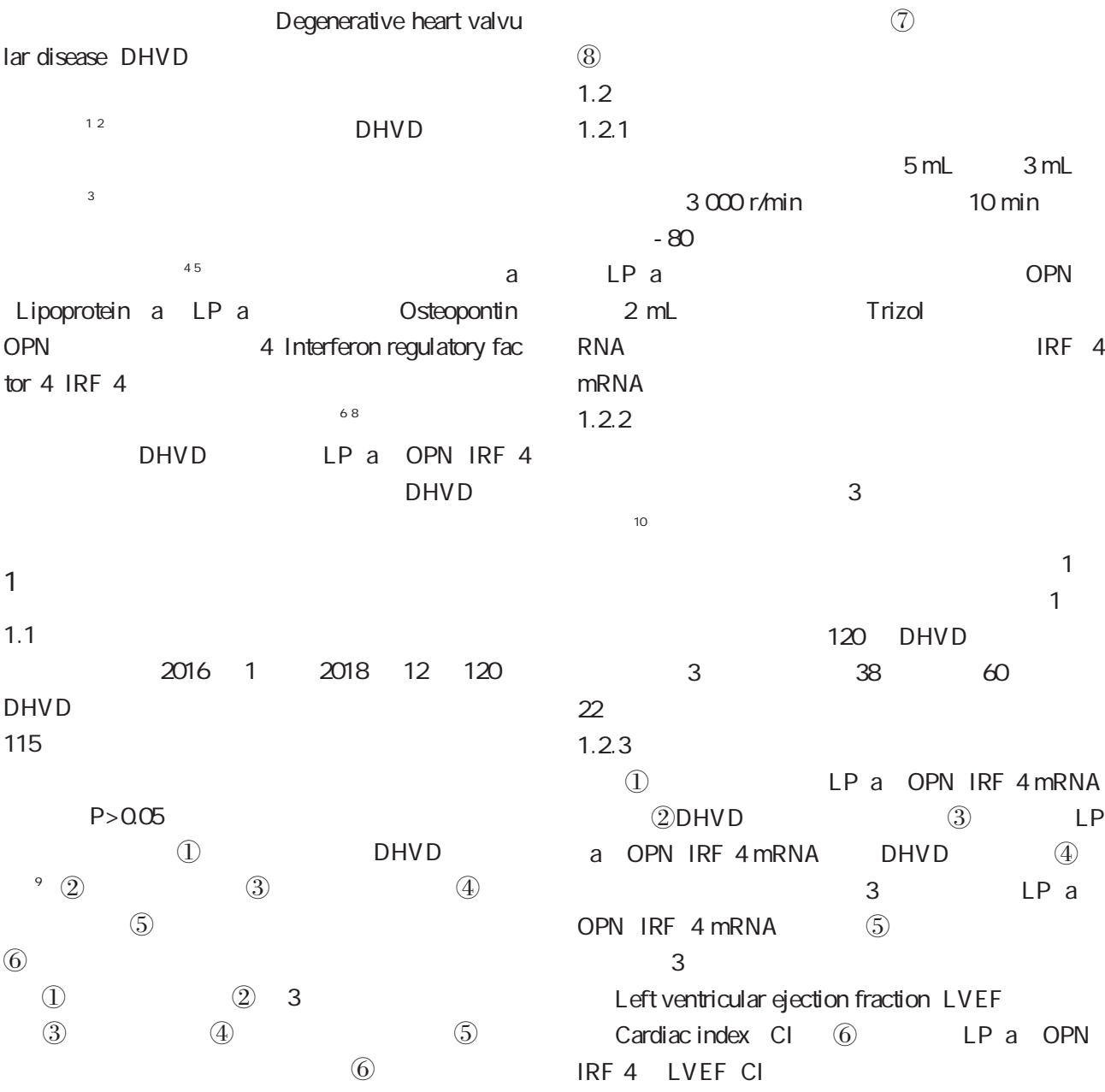
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history high blood pressure serum LP a OPN and IRF 4 mRNA are important influencing factors for the onset of DHVD P<0.05 . Coronary heart disease smoking history hypertension serum LP a OPN IRF 4 mRNA are important influencing factors of DHVD P<0.05 . Among the various serum indexes the area under the curve AUC for diagnosis of DHVD by LP a is the largest 0.802 the sensitivity of the cutoff value is 70.00% and the specificity is 86.09%. After the treatment for 3 months the serum LP a and OPN levels of the effective patients were lower than those of the effective and ineffective patients and the IRF 4 mRNA levels and LVEF CI were higher than those of the effective and ineffective P<0.05 . The serum LP a and OPN had negative correlation with LVEF and CI. Interestingly the IRF 4 mRNA had positive correlation with LVEF and CI P<0.05 . Conclusion The abnormal expression of serum LP a OPN and IRF 4 mRNA in DHVD patients is an important influencing factor of DHVD and has high application value in the diagnosis and evaluation of DHVD.

KEY WORDS Degenerative heart valve disease Lipoprotein a Osteopontin Interferon regulatory factor 4 Cardiac function Diagnosis



4 ROC

Table 4 ROC analysis results

	AUC	95%CI	Z	P		%	%
LP a	0.802	0.745-0.851	10.239	<0.001	>52.57 mg/dL	70.00	86.09
OPN	0.750	0.689-0.804	7.902	<0.001	>59.32 g/L μ	57.50	83.48
IRF 4 mRNA	0.758	0.699-0.812	8.295	<0.001	≤ 0.82	76.67	65.22

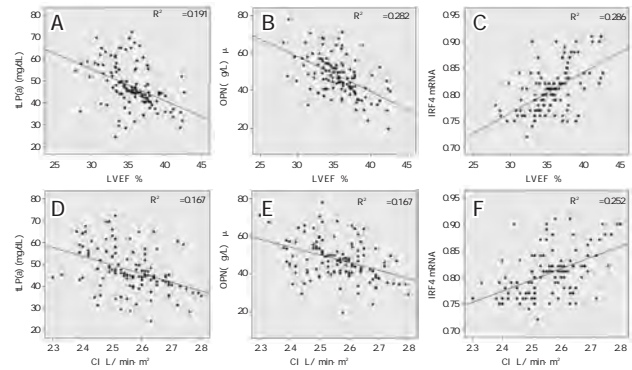
5 LP a OPN IRF 4 mRNA

$\bar{x} \pm s$

Table 5 Comparison of serum Lp a OPN and IRF 4 mRNA levels in patients with different therapeutic effects

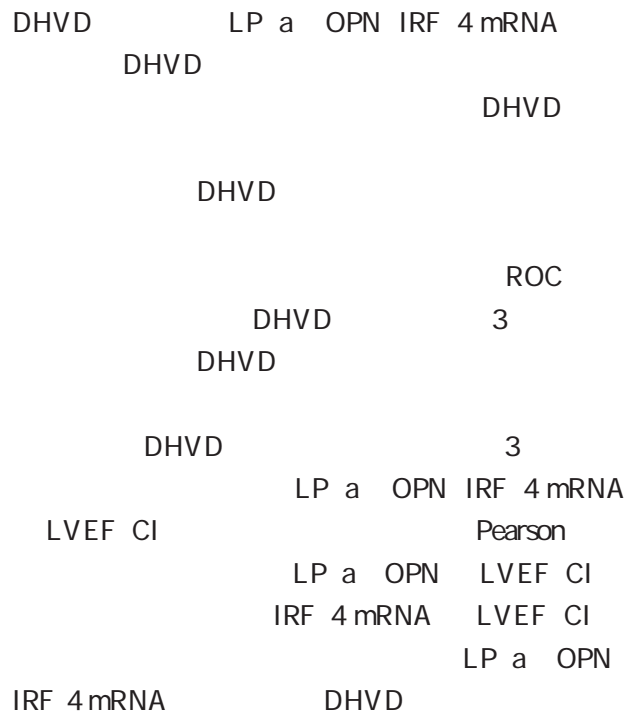
$\bar{x} \pm s$

	n	3
LP a mg/dL	38	42
	60	49
	22	54
F	0.226	15.220
P	0.798	<0.001
OPN g/L μ	38	40
	60	48
	22	58
F	0.092	31.731
P	0.912	<0.001
IRF 4 mRNA	38	0.83
	60	0.80
	22	0.77
F	1.648	25.242
LVEF %	38	38.40
	60	35.64
	22	33.29
F	0.522	24.542
P	0.595	<0.001
CI L/ min m^2	38	2.65
	60	2.56
	22	2.48
F	0.347	22.922
P	0.708	<0.001



A LP a LVEF B OPN LVEF
C IRF 4 mRNA LVEF D LP a CI
E OPN CI F IRF 4 mRNA CI

1
Figure 1 Serum indexes were correlated with cardiac function



12 IRF

T

IRF 4

13 14

Logistic mRNA DHVD LP a OPN IRF 4

LBP HSP70 DcR3

1* 2 1

LBP HSP70 DcR3

ARDS 2017 5 2019 5

92 ARDS 30d 64

28 LBP HSP70 DcR3 SOFA

II APACHE II 7

LBP DcR3 HSP70 SOFA APACHE II P<

Q05 7 P<Q05 LBP DcR3 SOFA

APACHE II HSP70 SOFA APACHE II P<Q05 7 HSP70AUC

Q.830 > 7 LBP Q.806 > 7 DcR3 Q.766 LBP HSP70 DcR3

P<Q05 LBP HSP70 DcR3 ARDS

ARDS

70 3 ROC

The value of serum LBP HSP70 DcR3 levels in predicting the outcome of patients with acute respiratory distress syndrome

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ABSTRACT Objective To investigate the value of serum lipopolysaccharide binding protein LBP heat shock protein 70 HSP70 and trap receptor 3 DcR3 levels in predicting the outcome of patients with acute respiratory distress syndrome ARDS . Methods 92 patients with ARDS admitted to our hospital from May 2017 to May 2019 were recruited as the research object. According to the prognosis of 30 days 64 surviving patients were selected as the survival group and 28 patients with all cause death were the death group. The serum levels of LBP HSP70 DcR3 sequential organ failure assessment sofa acute physiology and chronic health II APACHE II were compared between the two groups. Results The serum LBP and DcR3 levels were higher in the death group on the day of admission and the 7th day after admission than those in the survival group. HSP70 level was lower than that in the survival group. SOFA and APACHE II scores were higher than in the survival group P<0.05 . The difference in the survival group and the seventh day after admission was significant compared to the death group P<0.05 . The Serum LBP and DcR3 levels were positively correlated with SOFA and APACHE II scores and HSP70 was negatively corre

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lated with SOFA and APACHE II scores $P < 0.05$. Seventh day after admission HSP70 AUC 0.830 > LBP 0.806 > DcR3 0.766. There were significant differences in the survival curves of serum LBP HSP70 and DcR3 between the high risk groups and the low risk groups $P < 0.05$. Conclusion Serum LBP HSP70 and DcR3 levels are closely related to the severity of ARDS patients and are expected to be the main predictors of ARDS prognosis.

KEY WORDS Acute respiratory distress syndrome Lipopolysaccharide binding protein Heat shock protein 70 Trap receptor 3 ROC Lung injury

Acute respiratory dis 1.3
 tress syndrome ARDS 3 mL 12 min 3 000 r/
 ARDS 35.0% ~ min
 46.0%¹ ARDS DcR3 HSP70 LBP
 2
 Lipopolysacchoride Binding Pro 1.4
 tein LBP Toll 4 Toll like receptor ① LBP HSP70 DcR3
 TLR 4 ③ SOFA APACHE II SOFA
 70 Heat shock proteins 70 HSP70 6
 1-4
 3 Decoy receptor3 APACHE II 71 >17
 DcR3 ②
 LBP HSP70 DcR3 SOFA APACHE II
 ③ ARDS
 DcR3 ARDS ④ LBP HSP70 DcR3 ARDS
 ARDS ⑤ LBP HSP70 DcR3
 1.5
 SPSS 22.0
 1.1 $\bar{x} \pm s$ t n
 ARDS 2017 5 2019 5 92 % χ^2 Logistic
 64 30 d 28 Pearson
 P>0.05 ROC Kaplan Meier KM
 Log Rank P<0.05
 2
 1.2 ① ARDS ⑤ ② 2.1
 X BMI
 ③ ≤ 200 mmHg ④ ≤ 18 P>0.05 1
 mmHg ⑤ ⑥ LBP HSP70 DcR3
 ① 7 LBP DcR3
 ② HSP70 P<0.05
 ③ ④ 7
 P<0.05 2

1 n %

Table 1 Comparison of 2 groups of general data n %

	n=64	n=28	t/ χ^2	P
/	35/29	16/12	0.048	0.827
BMI kg/m ²	60.70±7.09	60.28±7.27	0.260	0.796
	20.92±1.86	21.14±2.05	0.506	0.614
	53 82.81	23 82.14	0.049	0.825
	5 7.81	2 7.14	0.100	0.752
	3 4.69	2 7.14	0.001	0.983
	3 4.69	1 3.57	0.097	0.754
ng/mL	7.40±1.08	6.83±1.86	1.847	0.068
	40/24	16/12	3.058	0.080

2 LBP HSP70 DcR3 $\bar{x} \pm s$

Table 2 Comparison of serum LBP HSP70 and DcR3 levels between 2 groups $\bar{x} \pm s$

	n	LBP g/mL μ	HSP70 g/L	DcR3 μ ng/mL
	28	121.90±32.27	1.02±0.31	3.16±0.62
	64	93.37±29.06	1.33±0.34	2.59±0.58
t		4.189	4.130	4.247
P		<0.001	<0.001	<0.001
	28	102.05±34.00	1.17±0.42	3.04±1.01
	64	67.44±22.46	1.85±0.69	1.83±0.60
7 t		5.774	4.829	7.149
P		<0.001	<0.001	<0.001
	28	19.85±3.23	0.15±0.06	0.12±0.04
	64	25.93±5.10	0.52±0.21	0.76±0.25
7 t		5.809	9.136	13.431
P		<0.001	<0.001	<0.001

2.3 SOFA APACHE II

7 SOFA APACHE II

P<0.05

7

P<0.05 3

2.4 LBP HSP70 DcR3 SOFA

APACHE II

Pearson

SOFA APACHE II

SOFA APACHE II

4

4 LBP HSP70 DcR3 SOFA APACHE II

Table 4 Correlation of serum LBP HSP70 DcR3 levels with sofa and Apache II scores

	LBP		HSP70		DcR3	
	r	P	r	P	r	P
SOFA	0.471	<0.001	0.446	<0.001	0.486	<0.001
7	0.583	<0.001	-0.529	<0.001	0.537	<0.001
APACHE II	0.503	<0.001	-0.512	<0.001	0.611	<0.001
7	0.621	<0.001	-0.635	<0.001	0.702	<0.001

2.5 ARDS

ARDS

MODS

DcR3 SOFA APACHE II

Logistic

MODS

DcR3 SOFA APACHE II

P<0.05 5

2.6 LBP HSP70 DcR3 ARDS

ROC

7

LBP HSP70 DcR3 ARDS

7 HSP70 AUC 0.830 > 7

LBP 0.806 > 7 DcR3 0.766

6 1

3 SOFA APACHE II $\bar{x} \pm s$

Table 3 Comparison of sofa and Apache II scores between 2 groups $\bar{x} \pm s$

	n	SOFA			APACHE II		
		7	7	7	7	7	7
	28	8.69±1.41	8.03±2.67	0.66±0.23	18.10±1.73	17.06±4.75	1.04±0.96
	64	7.37±1.35	5.39±1.80	1.98±0.30	16.96±1.40	12.34±3.22	4.62±1.13
t		4.258	5.550	20.744	3.340	5.562	14.605
P		<0.001	<0.001	<0.001	0.001	<0.001	<0.001

5 ARDS

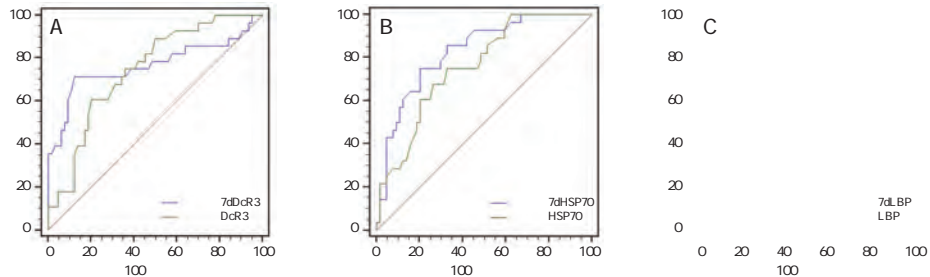
Table 5 Prognostic factors of death in patients with ARDS

	β	SE	Wald/ χ^2	OR	95%CI	P
MODS	1.553	0.420	13.670	4.725	2.711-8.235	<0.001
	1.426	0.517	7.605	7.105	2.437-7.105	<0.001
LBP	0.990	0.381	6.749	5.063	1.430-5.063	<0.001
HSP70	-0.615	0.273	5.082	0.907	0.322-0.907	0.013
DcR3	1.282	0.409	9.831	6.004	2.165-6.004	<0.001
SOFA	1.180	0.426	7.666	5.179	2.043-5.179	<0.001
APACHE II	0.973	0.312	9.723	4.081	1.715-4.081	<0.001

6 ROC

Table 6 ROC analysis results

	AUC	95%CI	Z		%	%	P
LBP	0.686	0.581-0.779	3.281	>100.30	78.57	59.38	0.001
HSP70	0.760	0.659-0.843	0.843	≤ 1.29	75.00	67.19	<0.001
DcR3	0.744	0.643-0.830	4.616	>3.02	60.71	79.69	<0.001
7 LBP	0.806	0.710-0.881	5.379	>92.28	67.86	90.62	<0.001
7 HSP70	0.830	0.737-0.900	7.413	≤ 1.45	75.00	79.69	<0.001
7 DcR3	0.766	0.667-0.848	4.095	>2.46	71.43	87.50	<0.001



A DcR3 ARDS ROC B HSP70 ARDS ROC C LBP ARDS ROC
1 ROC

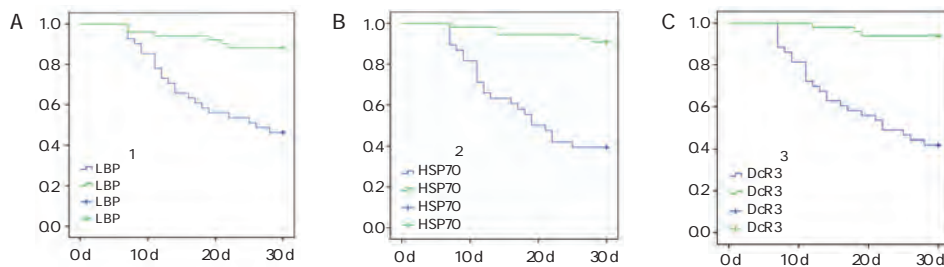
Figure 1 ROC curve

2.7

LBP HSP70 DcR3

= 0 = 1 ROC
KM

$\chi^2_1=19.438$ $\chi^2_2=31.134$ $\chi^2_3=30.894$ P<0.001 2



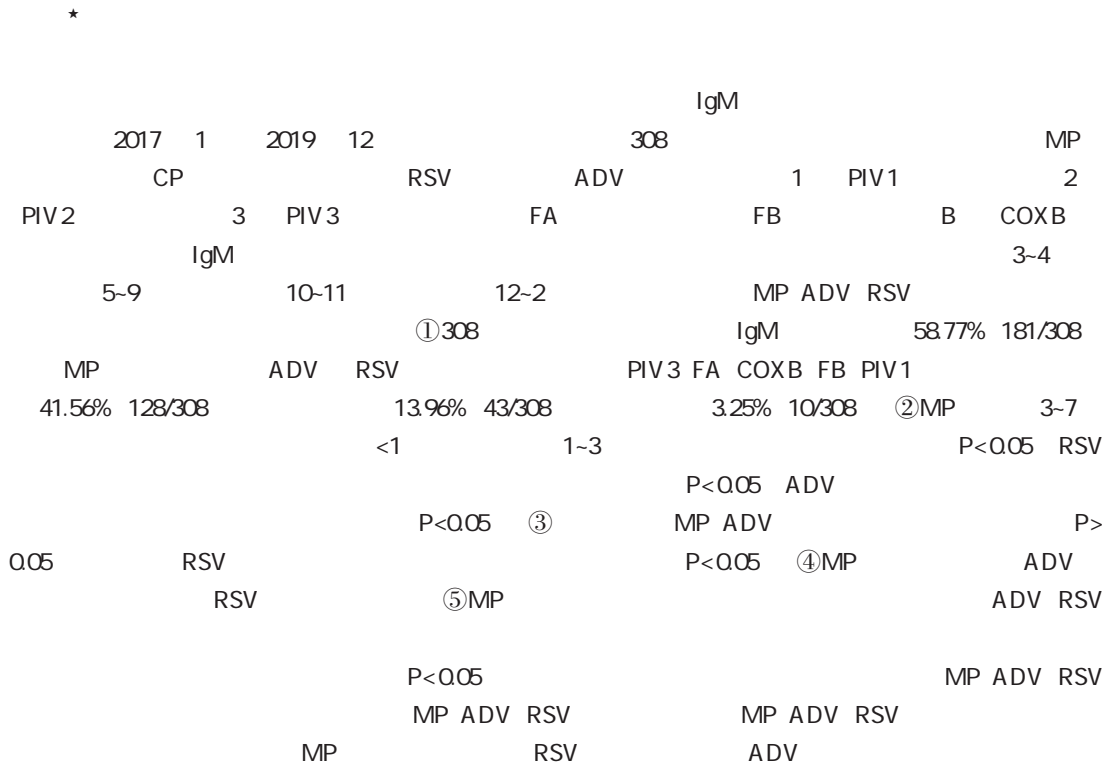
A LBP B HSP70 C DcR3

2 LBP HSP70 DcR3

Figure 2 Survival curve LBP HSP70 DcR3

3	ARDS		ARDS	
			DcR3	
			M2	
			ICAM 1 IL 8	
			Pearson	DcR3
			SOFA APACHE II	
^{6,7}	LBP I		DcR3	ARDS
	5-10 g/mL μ	LBP	DcR3	ARDS
	200 g/mL μ LBP	24 h		
LPS	A		LBP HSP70 DcR3	
	^{8,9}	ARDS	ARDS	ARDS
	7d	LBP	ARDS	ARDS
LPS		NF κB		
	α TNF α	6		
1	ICAM 1		1	Li H Zhou X Tan H et al. Neutrophil extracellular traps contribute to the pathogenesis of acid aspiration induced ALI/ARDS J . Oncotarget 2017 9 2 1772-1784.
	HSP70		2	
			3	J . 2016 22 6 487-491.
^{12,13}	HSP70	¹⁴	4	J . 2016 13 20 2916-2918.
	TNF α IL 1		5	J . 2016 16 6 409-411.
HSP70	ARDS		6	J . 2016 9 6 404-424.
		7	7	Peretti M Hervochon R Loi M et al. Predictors of post pneumonectomy respiratory failure and ARDS usefulness of normalized pulmonary artery diameter J . Intensive Care Med 2018 44 8 1357-1359.
		HSP70	8	J . 2016 21 1 15-21.
	T		9	J . 2015 21
TNF α		7	10	J . 2015
¹⁵	HSP70	ARDS	11	J . 2015
	HSP70			
	DcR3	33 kD		
	FasL			
	FasL/DcR3			
¹⁷	20	ARDS		
	DcR3			
DcR3	ARDS			
Cox	DcR3 ≥ 2.7 ng/mL	ARDS		
30 d				

IgM



Detection characteristics and epidemiological trend analysis of different pathogenic antigens and IgM antibodies in children with severe pneumonia

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Department of Special Needs Xiamen Children s Hospital Xiamen Fujian China 361006

ABSTRACT Objective To explore the detection characteristics and epidemic trend of IgM antibody anti different pathogens in children with severe pneumonia. Methods A total of 308 children with severe pneumonia admitted to Xiamen Children s Hospital from January 2017 to December 2019 were collected. Mycoplasma pneumoniae MP Chlamydia pneumoniae CP respiratory syncytial virus RSV and Virus ADV parainfluenza virus type 1 PIV1 parainfluenza virus type 2 PIV2 parainfluenza virus type 3 PIV3 influenza A virus FA influenza B virus FB Kosa Detection of multiple pathogenic

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antigens such as parvovirus B₁₉ COXB antigens and IgM antibodies were detected by immunofluorescence. According to the climatic characteristics of Xiamen the average temperature classification method is used to divide spring March to April summer May to September autumn 10 to November and winter 12 to February seasonal distribution characteristics of MP ADV and RSV infections were observed. The children with severe pneumonia and their complications were investigated. Results ① The positive rate of pathogenic IgM antibodies in 308 children with severe pneumonia was 58.77% 181/308 among which MP infection accounted for the highest proportion followed by ADV and RSV infection and others included PIV3 FA COXB FB PIV1. The single infection rate was 41.56% 128/308 the double infection rate was 13.96% 43/308 and the multiple infection rate was 3.25% 10/308. ② Among MP infections the infection rate is the highest between 3 and 7 years old preschool age which is significantly higher than <1 year infancy and 1 to 3 years infancy the difference is statistically significant $P < 0.05$ infants with RSV infection Period and early childhood are significantly higher than preschool age the difference is statistically significant $P < 0.05$. ADV infection in middle school and early childhood is significantly higher than infancy the difference is statistically significant $P < 0.05$. ③ The infection rates of MP and ADV in male and female children were not statistically significant $P > 0.05$. The RSV infection rates in male children were significantly higher than that in females and the difference was statistically significant $P < 0.05$. ④ MP infection is the highest in summer ADV infection is higher in spring and summer and RSV infection is the highest in summer. ⑤ Children with MP infection have higher rates of respiratory failure and myocardial damage complications. Various complications of children with ADV and RSV infection are also frequent. Children with multiple infections have complications such as respiratory failure pleural effusion myocardial damage and abnormal liver function significantly higher than that of a single pathogen $P < 0.05$. Conclusion The pathogens detected in children with severe pneumonia in our hospital are mainly MP ADV and RSV infections and the risk of double infection is high. Summer is the high risk season for MP ADV and RSV infection. MP ADV RSV infection is related to # wc sc pc iMP

Respiratory syncytial virus pneumonia RSV

Adenovirus ADV 1 Parainflu

enza virus type 1 PIV1 2 Parain

fluenza virus type 2 PIV2 3

Parainfluenza virus type 3 PIV3

influenza A FA influenza B

FB B Coxsackie b virus COXB

IgM

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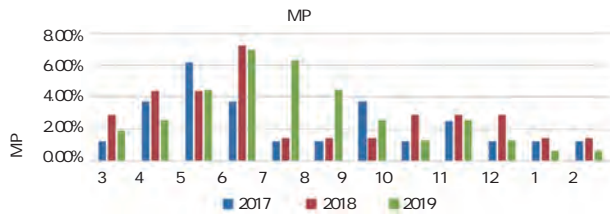


Figure 1 Severe pneumonia MP infection in different seasons

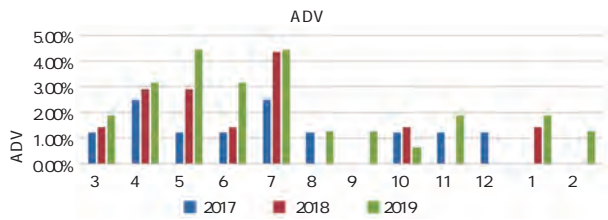


Figure 2 Severe Pneumonia ADV Infection in Different Season

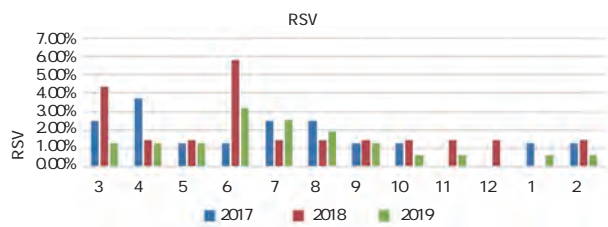


Figure 3 Severe pneumonia RSV infection in different seasons

2.5

RSV

P<0.05

4

3

8

Jayaweera

11

ADV

RSV

RSV

RSV

5.6

68%~89%

58%

ADV

RSV

MP

Jayaweera

MP

ADV

RSV

MP

MP

5-9 MP

MP

12 ADV

ADV

RSV

RSV

RSV

IgM

MP

MP

MP

MP

MP

MP

MP

MP

12 ADV

ADV

RSV

RSV

RSV

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RSV

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14

	n	
MP	103	0
ADV	62	
RSV	55	
	10	

χ^2

P

356 I™Ihí "Ó p I™'1ĀP 6öPI™IhÑ @ 6 I™Ihí "Ó p I™'Ö6 I™Ihí "Ó G TMT

		RSV			2019	J .
			15		2019 12 1	6 13
				5	. 2631	
	MP ADV RSV			6	J .	2018 18 6 746 749.
	MP	RSV			J .	2017 2018
	ADV			4	329 337.	2019 11
				7		
					J .	2017 24 6 455
					458	
		MP		8	. 2007- 2017	
					J .	2019 45 01 37 40.
16	MP			9	. 1629 7	
	MP				J .	2019 19 3 337 340.
				10	Jayaweera JAAS Noordeen F Kothalaweala S et al. A case series on common cold to severe bronchiolitis and pneumonia in children following human metapneumovirus infection in Sri Lanka J . BMC Research Notes 2018 11 1 127.	
	MP ADV RSV	ADV		11		
	MP	MP ADV RSV			J .	2017 38 12 1553 1556
	RSV	MP		12		2011 2017 5
	RSV	ADV			J .	
					2018 32 5 492 495.	
				13		
					J .	2018 45 4 712 714
1		. 2016			724.	
		J .		14		J .
	2018 10 3 148 152 173					
2		. 125			2017 55 8 580 585.	
	J .		2018 32 3 302	15		
	304.				J .	2014
3		. 2017	6		21 3 143 144.	
		J .	2019 45	16		
	01 48 51.				J .	2019
4					34 2 129 133.	

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13		HSPA12B				
		D .	2015 1 71.			
14		. HSP70 TLR NF κB		17	. DcR3	
		J .			J .	2017 42
	2015 35 3 406 411.				12 1644 1648.	
15		/				



SAA sTREM 1 CRP/PAB SP

*

A SAA 1 sTREM 1 C

CRP / PAB SP 82 80

21 61 1 3 5 7 d 1 3 5 7 d

SAA sTREM 1 ROC 3 SAA sTREM 1 CRP/PAB

SAA sTREM 1 CRP/PAB 3

3d SAA sTREM 1 CRP/PAB V SAA

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levels of SAA sTREM 1 and CRP/PAB in patients with severe infection and mild infection were compared and the relationship between the three and the degree of infection was analyzed. Results The serum SAA sTREM 1 CRP/PAB ratio reached a peak at 3 days postoperatively in the infected group and was higher than the uninfected group at 3 5 7 days postoperatively P<0.05 serum SAA was positively correlated with sTREM 1 and CRP/PAB ratio at the third days after surgery and sTREM 1 was positively correlated with CRP/PAB ratio P<0.01 the area under the ROC curve of serum SAA sTREM 1 CRP/PAB combined diagnosis of pulmonary infection after surgery was 0.876 serum SAA sTREM 1 and CRP/PAB ratios in patients with severe infection were higher than those in patients with mild infection at the third day after surgery P<0.05 serum SAA sTREM 1 and CRP/PAB ratios were positively correlated with the degree of infection P<0.05 . Conclusion The level of serum SAA sTREM 1 and the ratio of CRP/PAB were increased in SP patients with pulmonary infection after upper abdominal general anesthesia and it is closely related to the severity of infection and has a high diagnostic value in postoperative pulmonary infections which can provide theoretical basis for clinical diagnosis and disease evaluation.

KEY WORDS Amyloid A C reactive protein Prealbumin Schizophrenia Pulmonary infection

Schizophrenia SP	46.01±5.77	18-21 kg/m ²
SP	20.96±0.91 kg/m ²	SP
SP	①	②
SP	③	②
SP	①	
SP	③	
α ₂	1.2	
SP	1.2.1	
3	A	6-8 mL/kg
Serum amyloid A SAA	ViewT 8	Bene
1 Soluble triggering receptor expressed on myeloid cells 1 sTREM 1	A 2000xp	Aspect
	tral index BIS	Bispec
45	kg	10 min 1.0 g/μ
C	H 12070534	
C reactive protein CRP	0.4 g·kg ⁻¹ ·h ⁻¹	
Prealbumin PAB	0.05 mg/kg	3-5 g/kg μ
6	1.5-2.5 mg/kg	0.15 mg/kg
SAA sTREM 1	1-2L/min	8-10 mL/kg
SP	1 2	10-15 /min
	P _{ET} CO ₂ 35-40mmHg	
1	2-6 mg·kg ⁻¹ ·h ⁻¹	0.1-0.3 g·kg ⁻¹ ·h ⁻¹
1.1	0.1 mg·kg ⁻¹ ·h ⁻¹	1%~3%
2016 7		
2019 7		
SP 82	BIS 40-55	
	1.2.2	
61		
45	min	2 mL 15
35		SAA PAB
31-61		

sTREM 1 P>

0.05 1

1.3

① 1 3 5 7 d SAA
 sTREM 1 CRP/PAB ② 3 d
 SAA sTREM 1 CRP/PAB ③
 3 d SAA sTREM 1 CRP/PAB
 ④
 SAA sTREM 1 CRP/PAB

Table 1 Comparison of basic in 2 groups

	n	%	$\bar{x} \pm s$	χ^2/t	P
n=21	n=61				
40.31±5.33	41.10±5.16		0.600	0.550	
/	12/9	34/27	0.013	0.911	
			0.010	0.919	
13 61.90	37 60.66				
8 38.10	24 39.34				
21.46±1.33	20.96±1.22		1.583	0.117	
kg/m ²					
h	2.49±0.46	2.53±0.50	0.322	0.748	
min	133.12±31.64	129.87±29.55	0.427	0.671	

sTREM 1 CRP/PAB

1.4

SPSS 22.0 n
 % χ^2 $\bar{x} \pm s$
 t Pearson 3
 d SAA sTREM 1 CRP/PAB
 P<0.05

2.2 SAA sTREM 1 CRP/PAB
 1 d SAA sTREM 1 CRP/PAB
 P>0.05

2

3 d SAA sTREM 1 CRP/PAB

2.1

3 5 7 d P<0.05

2

Table 2 Serum SAA sTREM 1 and CRP/PAB ratios

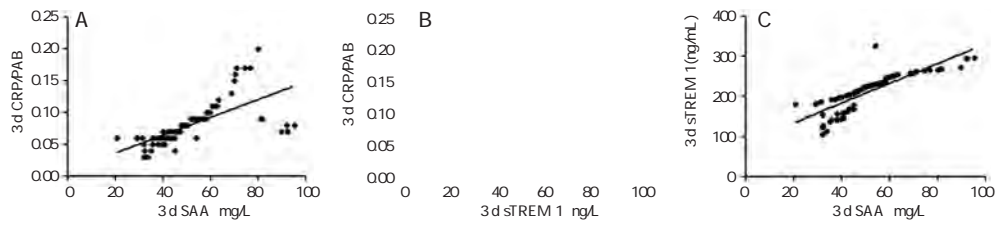
	n	1 d	3 d	5 d	7 d
SAA mg/L	21	35.42±6.28	64.11±21.37	51.04±15.24	40.78±10.07
	61	37.27±8.41	40.06±13.30	24.07±7.69	13.71±4.10
t		0.922	5.899	10.533	17.366
P		0.359	<0.001	<0.001	<0.001
sTREM 1 ng/mL	21	206.29±57.41	267.53±60.04	218.49±50.71	170.56±41.25
	61	202.44±52.09	210.14±53.30	174.06±38.46	132.02±29.14
t		0.285	4.120	4.195	4.674
P		0.777	<0.001	<0.001	<0.001
CRP/PAB	21	0.10±0.04	0.30±0.10	0.23±0.07	0.15±0.04
	61	0.09±0.03	0.12±0.04	0.07±0.02	0.04±0.01
t		1.206	11.696	16.194	19.949
P		0.232	<0.001	<0.001	<0.001

2.3 3 d SAA sTREM 1 CRP/PAB

2.4 3 d SAA sTREM 1 CRP/PAB

Pearson 3 d SAA $r_1=0.845$
 $r_2=0.671$ sTREM 1 CRP/PAB
 sTREM 1 $r=0.784$ CRP/PAB
 0.01 1

SAA sTREM 1 CRP/PAB
 AUC 0.876 SAA 0.768
 sTREM 1 0.701 CRP/PAB 0.786 P<0.01 3
 2



A SAA CRP/PAB B sTREM 1 CRP/PAB C SAA sTREM 1
1 3d SAA sTREM 1 CRP/PAB

Figure 1 Correlation between serum SAA sTREM 1 and CRP/PAB ratios at 3 days after surgery

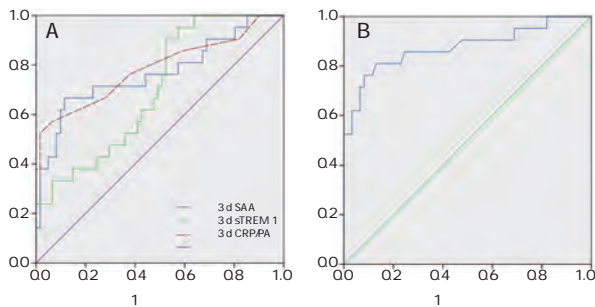
3
Table 3 Diagnostic value

	AUC	95%CI	%	%
SAA	0.768	0.662-0.854	>58.4	66.67
sTREM 1	0.701	0.590-0.797	>210.3	90.48
CRP/PAB	0.786	0.682-0.869	>0.1	52.38
	0.876	0.785-0.939	-	76.19

4
SAA sTREM 1 CRP/PAB
 $\bar{x} \pm s$

Table 4 Comparison of serum SAA sTREM 1 CRP/PAB ratios in patients with different infection $\bar{x} \pm s$

	n=8	n=13	t	P
SAA mg/L	73.96±17.03	58.05±13.29	2.396	0.027
sTREM 1 ng/mL	288.85±40.16	254.41±31.22	2.204	0.040
CRP/PAB	0.35±0.07	0.27±0.04	3.355	0.003



A SAA sTREM 1 CRP/PAB ROC
B SAA sTREM 1 CRP/PAB ROC

2 ROC
Figure 2 ROC curve

2.5 SAA sTREM 1
CRP/PAB

3 d

8

13

SAA sTREM 1

CRP/PAB

P<0.05

4

2.6 SAA sTREM 1 CRP/PAB

Spearman

SAA r=0.512

sTREM 1 r=0.0473 CRP/PAB r=0.608

P<

3 d

SAA

0.05

3

8 9

20% SP

10

SAA

11

12

SAA

SAA

1 d

SAA

SAA mRNA

SAA

T

IL 17 IL 23

13

SAA

SAA N
ty lipoprotein HDL
A2l ApoA2l

High densi

HDL
SAA/HDL

Cells 1 TREM 1 Dampens Proinflammatory Cytokine Se
cretion by Lamina Propria Cells from Patients with IBD J .
Inflamm Bowel Dis 2016 22 8 1803 1811.

6

2~3

SAA ¹⁴

CRP/ALB
J . 2016
23 6 630 632

SAA

7

Korean Neuropsychiatric Association. Korean Guidelines for
the Pharmacological Treatment of Social Anxiety Disorder
Initial Treatment Strategies

¹⁵

SAA

sTREM 1 TREM 1

^{16 17}

sTREM 1

PAB

CRP

SAA sTREM 1 CRP/

PAB

SP

SAA sTREM 1 CRP/PAB

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TSP 1 PDGF

1* 2

1 TSP 1 PDGF
 2017 3 2019 2 65 ICH
 Rankin n=37
 Logistic
 90 d 5 n=23
 NIHSS P<0.05
 Logistic OR=1.275 95%CI 1.052-1.545 OR=1.551 95%CI 1.219-1.974
 OR=1.368 95%CI 1.124-1.664 NIHSS OR=2.537 95%CI 1.109-5.802
 TSP 1 OR=2.659 95%CI 1.202-5.881 PDGF OR=3.086 95%CI 1.324-7.197 6
 P<0.05 ROC TSP 1 121.23 ng/mL
 78.26% 78.38% AUC 0.877 95%CI 0.782-0.971 PDGF
 369.34 ng/L 73.91% 70.27% AUC 0.778 95%CI 0.638-0.918
 0.881-1.000 86.96% 67.57% AUC 0.943 95%CI
 NIHSS TSP 1 PDGF
 TSP 1 PDGF
 1

Clinical value of serum TSP-1 and PDGF in evaluating short-term prognosis in patients with intracerebral hemorrhage

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2. Department of Neurology Bayannaer Hospital Bayannaer Inner Mongolia 015000

ABSTRACT Objective To study the clinical value of serum TSP 1 and PDGF in evaluating short term prognosis in patients with intracerebral hemorrhage. Methods 65 cases of patients with intracerebral hemorrhage ICH from March 2017 to February 2019 in our hospital were retrospectively analyzed. After 90 days of follow up 5 patients withdrew from the study because of their personal reasons. The patients were divided into favorable outcome group n=37 and unfavorable outcome group n=23 depending on modified Rankin scale scores. The basic information and treatment of patients in the two groups were compared using single factor analysis and the significantly different single factors were analyzed by non conditional logistic regression analysis. Finally the dangerous factors for poor prognosis were established. Results There were significant differences in body mass index hypertension hematoma volume NIHSS score pulmonary

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infection broken ventricle and fasting blood glucose between the two groups. Logistic regression analysis show that hypertension OR=1.275 95%CI 1.052-1.545 fasting blood glucose OR=1.551 95%CI 1.219-1.974 hematoma volume OR=1.368 95% CI 1.124-1.664 NIHSS score OR=2.537 95% CI 1.109-5.802 TSP 1 OR=2.659 95% CI 1.202-5.881 and PDGF OR=3.086 95% CI 1.324-7.197 are

1.6

SPSS 25.0

$x \pm s$

t

n %

χ^2

Logistic

P<

0.05

2

2.1

NIHSS

P<0.05

1

2.2

TSP 1

PDGF

TSP 1 PDGF

P<0.05

2

2.3 Logistic

90d

Logistic

Logistic

NIHSS

TSP 1

PDGF 6

P<0.05

3

2.4

TSP 1

PDGF

X

1

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ROC

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ROC

TSP 1

121.23 ng/

mL PDGF

369.34 ng/L

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ICH

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ICH^a

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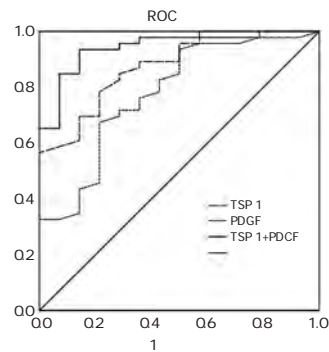
Table 2 Comparison of serum TSP 1 PDGF value between the 2 groups $\bar{x} \pm s$

	n	TSP 1 ng/mL	PDGF ng/L
	37	102.47±18.65	341.34±32.59
	23	165.34±10.76	421.32±52.60
t		16.548	8.205
P		<0.001	<0.001

3 Logistic

Table 3 Result of logistic regression

	B	S.E	Wald	P	OR	95%CI
	0.243	0.098	6.148	0.013	1.275	1.052-1.545
	0.439	0.123	12.739	<0.001	1.551	1.219-1.974
	0.313	0.100	9.797	0.002	1.368	1.124-1.664
NIHSS	0.931	0.422	4.867	0.027	2.537	1.109-5.802
TSP 1	0.978	0.405	5.831	0.016	2.659	1.202-5.881
PDGF	1.127	0.432	6.806	0.009	3.086	1.324-7.197
	-0.564	0.121	21.726	<0.001	-	-



1 Logistic ROC
Figure 1 ROC curve of the prediction model based on Logistic regression analysis

Logistics
NIHSS ICH

14

15

16

ICH
NIHSS
ICH
ICH

17

TSP 1

18

ICH TSP 1

19

PDGF

α

PDGF

PDGF

20

ICH

TSP 1 PDGF

ICH

NIHSS TSP 1 PDGF

PDGF TSP 1

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LP a OPN IRF 4 mRNA
 DHVD DHVD
 DHVD
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 40 8 18 22.
 14 4
 J . 2016 22
 5 392 397.

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TNF α PCT IL 1 β

1* 2 2

PCT 1 β IL 1 β α TNF α
80 2015 1 2017 12
160
TNF α PCT IL 1 β 3
1 TNF α PCT IL 1 β
P>0.05 1 3 5 TNF α PCT IL 1 β "

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* E-mail 545089160@qq.com

TNF α PCT IL 1 β 1 5 1 3 5 TNF α PCT IL 1 β
 1 3 5
 TNF α PCT IL 1 β P<
 P>0.05 0.05 1

Table 1 Comparison of dynamic trends of serum TNF α PCT IL 1 β in two groups of women $\bar{x} \pm s$

	TNF α ng/mL			PCT g/mL μ			IL 1 β pg/mL		
	1	3	5	1	3d	5	1	3	5
	1.55 \pm 0.32	2.67 \pm 0.69	3.04 \pm 0.55	0.81 \pm 0.27	2.23 \pm 0.69	2.52 \pm 0.77	0.81 \pm 0.27	2.23 \pm 0.69	2.52 \pm 0.77
	1.48 \pm 0.29	1.50 \pm 0.31	1.44 \pm 0.38	0.78 \pm 0.24	0.63 \pm 0.20	0.47 \pm 0.15	0.78 \pm 0.24	0.63 \pm 0.20	0.47 \pm 0.15
t	1.702	18.125	26.334	0.875	27.184	32.528	0.875	27.184	32.528
P	0.090	0.000	0.000	0.382	0.000	0.000	0.382	0.000	0.000

2.2 3 TNF α PCT IL 1 β 7
 ROC 3 TNF α PCT IL 1 β 5%
 PCT TNF α IL 1 β 8
 AUC 0.888
 0.766 0.751 1.26 g/ μ
 mL 2.00 ng/mL 2.51 pg/mL P<0.05 1 C 6
 C

40%
9

Tumor Necrosis Factor TNF

TNF α TNF β

TNF α

TNF α

T

Ts

PCT

PCT

PCT

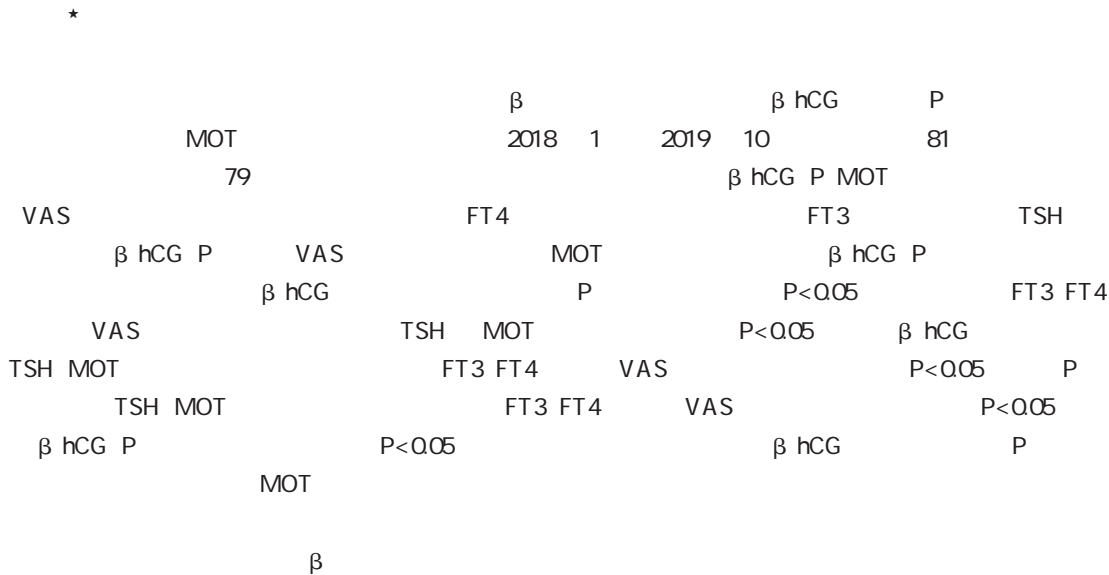
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β hCG



Correlation of serum β - hCG and progesterone expression with thyroid function and motilin in patients with hyperemesis gravidarum

LI Feng* LU Haifeng CUI Bingyi QIN Chunyi

Clinical laboratory Tangshan hospital of traditional Chinese medicine Tangshan Hebei China 063000

ABSTRACT Objective To investigate the expression of serum β human chorionic gonadotropin β hCG and progesterone P in patients with hyperemesis gravidarum and the correlation with thyroid function and motilin MOT . Methods From January 2018 to October 2019 81 pregnant women with severe vomiting in hospital were selected as the study group and 79 normal pregnant women were selected as the control group. The serum β hCG P MOT levels hyperemesis gravidarum VAS score thyroid function free thyroxine FT4 free triiodothyronine FT3 and thyroid stimulating hormone TSH were measured and compared between the two groups. The correlations of serum β hCG and P levels with VAS score and thyroid function and MOT were analyzed. Results The serum β hCG level in the study group was higher than that in the control group and the serum P level was lower than that in the control group P<0.05 . Serum FT3 and FT4 levels and VAS scores in the study group were higher than those in the control group and serum TSH and MOT levels were lower than those in the control group P<0.05 . There was a significant negative correlation between serum β hCG levels and serum TSH and MOT levels and a significant positive correlation with se

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2

2.1

P>0.05

2.2

β hCG P

FT₄
22 23
β hCG
FT₃ FT₄
TSH MOT

24 25
β hCG P
MOT TSH FT₃ FT₄
β hCG P

β hCG P
MOT

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CysC α1 MG β2 MG

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C CysC α1 α1 MG β2 β2 MG

COPD 2013 1

2016 8 206 COPD SPO2 64 86

56 50 4 CysC α1 MG β2 MG Scr

BUN ROC CysC α1 MG β2 MG COPD COPD

Pearson CysC BUN α1 MG β2 MG Scr ① COPD

CysC α1 MG β2 MG COPD Scr BUN P>0.05

②Cys C P<0.05 α1 MG β2 MG

P<0.05 CysC α1 MG β2 MG P<0.05

CysC P<0.05 ③α1 MG β2 MG CysC Scr P<0.05

④ROC CysC α1 MG β2 MG COPD 0.810 0.743 0.812 0.915

0.936 1.74 mg/L 12.22 mg/L 2.34 mg/L 0.806 0.726 0.794 0.903

0.795 0.705 0.782 0.903 CysC α1 MG β2 MG 0.864 0.832 0.812

1.62 mg/L 12.08 mg/L 2.58 mg/L 0.824 0.816 0.808 0.914

0.786 0.774 0.762 0.903 CysC α1 MG β2 MG COPD

C α1 β2

The detection and significance of CysC α1-MG β2-MG of early kidney injure in patients with chronic obstructive pulmonary disease

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Dongying New District Hospital Dongying Shandong China 257000

ABSTRACT Objective To investigate the detection value of serum cystatin C CysC α1 microglobulin α1 MG and β2 microglobulin β2 MG in chronic obstructive pulmonary disease COPD and correlation of the early stage of hypoxia kidney injury. Methods A total of 206 elderly patients with COPD from January 2013 to August 2016 in our hospital were selected as the study object. Based on the blood gas analysis they were divided into COPD with mild moderate and severe hypoxia groups. A total of 50 healthy people were selected as control group. The CysC α1 MG β2 MG Scr levels were detected and compared for analysis. The ROC curve was used to analyze the diagnostic value of CysC α1 MG and β2 MG on COPD and COPD combined with renal injury. Pearson correlation analysis was used to analyze the correlation between CysC BUN α1 MG and β2 MG and Scr. Results ①Compared with control group CysC α1

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MG β2 MG levels in COPD group significantly increased and the Scr levels had no statistical significance on the difference with the control group $P > 0.05$. ② CysC level in mild moderate and severe hypoxia group were significantly higher than that in control group and the differences had no statistical significance $P < 0.05$ α1 MG β2 MG levels in moderate and severe hypoxia group were significantly higher than that in control group $P < 0.05$ α

AUC COPD P<0.05 2.2 4 CysC α1 MG β2 MG Scr BUN CysC P>0.05 1 α1 MG β2 MG P<0.05 2.1 COPD CysC α1 MG β2 MG CysC α1 MG β2 MG Scr BUN P<0.05 COPD CysC α1 MG β2 MG Scr BUN P<0.05 CysC P<0.05 2

Table 1 Comparison of related indicators between the 2 groups $\bar{x} \pm s$

	n	Scr mol/L μ	BUN mmol/L	CysC mg/L	α1 MG mg/L	β2 MG mg/L
COPD	50	76.01±15.76	5.84±1.43	1.14±0.22	8.84±1.56	1.64±0.26
t	206	83.36±34.39	6.43±3.12	1.69±0.53	13.63±4.35	2.63±1.15
P		1.318	1.562	4.472	5.124	4.115
		0.231	0.128	0.013	0.004	0.021

Table 2 Comparison of related indicators between the 4 groups $\bar{x} \pm s$

	n	Scr mol/L μ	BUN mmol/L	CysC mg/L	α1 MG mg/L	β2 MG mg/L
	50	76.01±15.76	5.84±1.43	1.14±0.22	8.84±1.56	1.64±0.26
	64	80.58±17.43	6.36±1.61	1.37±0.41 ^a	9.54±1.85	1.93±1.35
	86	86.56±19.23	6.73±1.98	1.64±0.72 ^{ab}	12.24±1.81 ^a	2.64±1.81 ^a
	86	93.38±21.73	7.04±2.09	1.98±1.23 ^{abc}	17.42±2.79 ^b	3.72±2.79 ^b
F		9.677	6.379	7.240	8.660	9.275
P		0.000	0.001	0.000	0.000	0.000

2.3 CysC α1 MG β2 MG COPD ROC
 ROC CysC COPD
 0.810 1.74 mg/L
 COPD 0.806
 0.795 α1 MG 0.743
 12.22 mg/L COPD
 0.726 0.705 β2 MG
 0.812 2.34 mg/L
 COPD 0.794
 0.782 3 0.915
 0.903 0.897 1

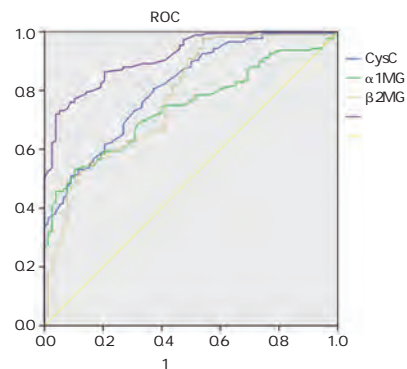


Figure 1 ROC curves of CysC α1 MG β2 MG for COPD prediction

2.4 CysC α1 MG β2 MG BUN Scr
 Pearson CysC r=0.672 P=0.011
 α1 MG r=0.415 P=0.031 β2 MG r=0.550 P=0.029
 BUN r=0.023 P=0.436 COPD
 Scr P<0.05 0.856 1.62 mg/L COPD
 0.824 0.786 α1 MG

2.5 CysC α1 MG β2 MG COPD
 ROC
 ROC CysC
 1.62 mg/L COPD
 0.824 0.786 α1 MG

	0.722	12.08 mg/L			COPD	CysC α1 MG β2
COPD		0.816	MG		Scr	Scr
0.774 β2 MG		0.812				
2.58 mg/L	COPD			Scr		CysC α1 MG β2
0.808	0.762		MG		Scr	
0.936	0.914	0.903	2		¹³	
						CysC α1 MG
			β2 MG			Ομ V5fĐ 0 CV fÙf 8E4(DôĐ @ q 4 . (

3

COPD

8

COPD

9 10

CysC

2

CysC COPD

COPD

α1 MG

β2 MG

COPD

COPD

Scr

CysC α1 MG β2 MG

after treatment they were divided into complete remission group n=48 and incomplete remission group n=32. The levels of COX-2, VEGF, p53 protein in the two groups were analyzed. The incidence of central nervous system leukemia (CNSL) between the two groups was compared. The predictive value of COX-2, VEGF and p53 protein in the prognosis of AML was analyzed using the receiver operating characteristic curve (ROC). Results: The levels of COX-2, VEGF and p53 proteins in the study group were significantly higher than those in the control group and the difference was statistically significant (P<0.05). However, after treatment, the levels of COX-2, VEGF and p53 protein in the study group were significantly lower than those before treatment. The difference was statistically significant (P<0.05). After treatment, the levels of humoral immune indicators IgA, IgG and IgM in the study group were significantly lower than before treatment (P<0.05). After treatment, the cell immune indicators of CD3+, CD4+, CD8+ and CD4+/CD8+ in the study group were significantly lower than before treatment (P<0.05). Correlation analysis indicates that there is a significant positive correlation between COX-2, VEGF and p53 proteins (P<0.05). The difference in COX-2, VEGF and p53 protein levels between AML patients in complete remission group and incomplete remission group was statistically significant (P<0.05). The difference in median survival between AML patients in complete remission group and incomplete remission group was statistically significant (P<0.05). ROC curve results showed that the area under the curve (AUC) of COX-2, VEGF, p53 protein and combined detection in CNSL diagnosis were 0.836 [95%CI 0.776-0.896], 0.802 [95%CI 0.730-0.873], 0.716 [95%CI 0.639-0.793] and 0.905 [95%CI 0.863-0.947]. Conclusion: The levels of serum COX-2, VEGF and p53 are of great significance for the clinical treatment effect and prognosis evaluation of elderly AML patients.

KEY WORDS: Acute myeloid leukemia, Old age, Cyclooxygenase 2, Vascular endothelial growth factor, p53 protein

acute myeloid leukemia

AML / 6 COX-2 VEGF p53
AML 1 65 AML
AML 10
AML 1
+ + 1.1
Ara c+ACR+G CSF CAG 2014 1 2019 1 80
DNA AML AML 49 31
60-76 68.8±2.4 M1 8 M2
14 M4 18 M5 31 M6 9
2 CAG 40
24 16 60-78 68.3±
COX-2 2 Cyclooxygenase 2 25 P>0.05
3 ①
vascular endothelial growth fac AML 7 ②
tor VEGF ③ ≥60 ④ AML MICM
VEGF M1 M2 M4 M5 M6 ⑤
45 P53 ① ②
p53

④ ③ ⑤ T
 CD3+ CD4+ CD8+ CD4+/CD8+
 1.2 1.3.3 1~5
 AML CAG central nervous system leukemia CNSL
 15mg/m² d d1 d5 20mg/d d1 d4 10mg/ CNSL
 m² q12h d1 d14 2016⁸
 150 g q12h d1 d14 >20×10⁹/L 1.4
 SPSS 20.0
 % χ^2
 1.3 $\bar{x} \pm s$ t Spearman
 1.3.1 COX 2 VEGF p53 COX 2 VEGF p53
 5 mL receiver operator characteristic curve ROC
 30 min 3 000 r/min 15 min COX 2 VEGF p53 CNSL
 enzyme linked P<0.05
 immunosorbent assay ELISA COX 2 VEGF
 p53 2
 AML 14 d 5 mL 2.1
 COX 2 VEGF p53
 1.3.2 P<0.05
 14 d P<0.05
 IgA IgG IgM
 P<0.05
 CyFlow Partec 1
 1 $\bar{x} \pm s$

Table 1 Changes of serum indicators in the 2 groups $\bar{x} \pm s$

	n=80		n=40	t	P
COX 2 ng/L	53.2±6.1	31.0±3.1	30.3±3.2 ^a	29.019	0.000
VEGF pg/mL	158.8±35.7	72.2±7.5	70.7±6.9 ^a	21.233	0.000
p53 ng/L	211.5±33.8	108.3±13.7	106.7±14.9 ^a	25.309	0.000
IgA g/L	2.3±0.9	1.5±0.7	-	4.276	0.000
IgG g/L	11.7±1.9	9.1±1.6	-	9.362	0.000
IgM g/L	1.0±0.2	0.7±0.2	-	9.487	0.000
CD3+ %	54.2±6.6	38.7±4.5	-	17.355	0.000
CD4+ %	34.2±4.3	28.8±3.2	-	9.011	0.000
CD8+ %	29.0±2.7	20.9±2.6	-	19.328	0.000
CD4+/CD8+	1.2±0.3	0.9±0.2	-	7.442	0.000

^aP<0.05

2.2 COX 2 VEGF p53 P<0.05

Table 2 Correlation analysis of COX 2 VEGF and p53 proteins with various indicators

	COX 2		VEGF		p53	
	r	P	r	P	r	P
COX 2	-	-	0.583	0.000	0.612	0.000
VEGF	0.501	0.000	-	-	-	0.000
p53	0.559	0.000	0.537	0.000	-	-

2.3 AML COX 2 VEGF Overall Survival OS P<0.05 AML OS P<

Table 3 Comparison of various indicators in different treatment outcomes of elderly AML patients

	n	COX 2 ng/L	VEGF pg/mL	p53 ng/L	OS
	48	31.5±4.4	70.3±7.7	102.3±10.4	12.6±2.7
	32	44.1±5.3	166.9±20.8	198.7±10.8	5.9±1.6
t		11.555	25.148	39.997	13.914
P		0.000	0.000	0.000	0.000

2.4 COX 2 VEGF p53 CNSL ROC CNSL ROC AUC AML

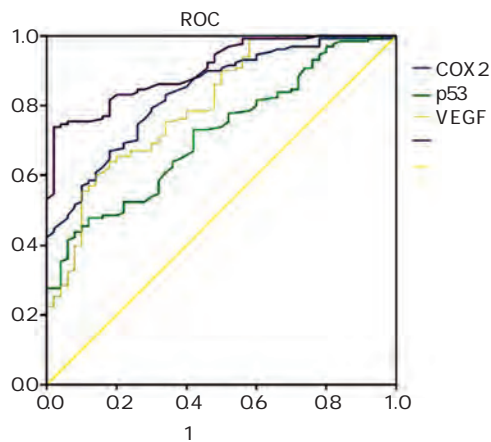


Figure 1 COX 2 VEGF and p53 predict ROC curves of CNSL

AML GO S DNA DNA AML COX 2 COX 2 DNA

13¹⁴ COX 2
 VEGF
 AML J . Cancer Res 2019 79 6 1165 1177.

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 VEGF
 VEGF
 J . 2018 38 24 5936

5938

6 p53
 p53
 P P53
 J . 2017 35 2

7 DNA
 p53
 425 428
 M . 3 .
 2007 110

16¹⁷ CAG
 VEGF p53
 AML COX 2
 8
 2016 J . 2016 37

10 837 845.

9 VEGF p53
 AML
 9
 J .

10 AML
 2019 27 5 1431 1435.

11 COX 2 VEGF p53
 COX 2 VEGF p53
 OS
 11
 2019 39 20 4931 4934.

12 AML
 COX 2 VEGF p53
 ROC
 CNSL
 COX 2
 AUC 3
 VEGF p53
 AML
 CNSL
 COX 2 VEGF p53
 AML
 12
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5480

2 /
 J . 2019 27 2

390 395.

3 Carter BZ Mak PY Wang X et al. An ARC regulated IL1 β /Cox 2/PGE2 β catenin/ARC circuit controls leukemia microenvironment interactions and confers drug resistance in



CD40 CD40L BAFF ITP

1* 2 1 1 1 1 1

CD40 CD40L BAFF ITP

ITP 80 BAFF ITP

100

CD40 CD40L BAFF ITP

2 CD40 CD40L BAFF

CD40 CD40L BAFF CD40 CD40L

BAFF P<0.05 CD40 CD40L

BAFF P<0.05 ITP CD40 CD40L BAFF

r<0 P<0.05 ROC CD40 CD40L BAFF ITP

AUC>0.7 P<0.05 ITP CD40 CD40L ITP

CD40 CD40L BAFF ITP

The predictive value of peripheral blood CD40 CD40L expression and serum BAFF level on the efficacy of patients with immune thrombocytopenic purpura

YIN Fenglei^{1*} YIN Juan² ZHAO Fang¹ ZHANG Rui¹ LIU Jing¹ ZHANG Wei¹ LI Shuchen¹ WANG Juan¹

1. Department of Hematology Cangzhou Central Hospital Cangzhou Hebei China 061000 2. Operating Room of Cangzhou People s Hospital Cangzhou Hebei China 061000

ABSTRACT Objective To investigate the predictive value of peripheral blood CD40 CD40L expression and serum BAFF level in the treatment of immune thrombocytopenic purpura ITP patients. Methods 80 patients with ITP admitted to our hospital were recruited as the observation group. 100 healthy people who underwent physical examination in our hospital were selected as the control group. Peripheral blood CD40 CD40L expression and serum BAFF level were observed in both groups. The patients were divided into two groups according to the results of efficacy judgment. The expression of CD40 CD40L and serum BAFF in peripheral blood from the two groups were compared. Results The expression of CD40 CD40L and serum BAFF in the peripheral blood in the observation group were significantly higher than those in the control group P<0.05 . The expression of CD40 CD40L and serum BAFF in peripheral blood of patients with non re

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response group were significantly higher than those in response group $P < 0.05$. The expression of CD40, CD40L and serum BAFF in peripheral blood of patients with ITP were negatively correlated with curative effect $r < 0$, $P < 0.05$. ROC curve analysis results show that the peripheral blood CD40, CD40L and BAFF have high predictive value for ITP efficacy, all $AUC > 0.7$, $P < 0.05$. Conclusion: The peripheral blood CD40 and CD40L in patients with ITP have a high predictive value for the efficacy of ITP patients.

KEY WORDS: Bone marrow CD40, CD40L, Serum BAFF level, ITP, Curative effect

idiopathic thrombocytopenic purpura (ITP)

1.2

① CD40 CD40L

4 mL

CD40 FITC CD40L PE

10 L μ R&G

PBS

Attune NxT

10 000

② BAFF 2 mL

3 000 r/min 15 min 0.8 mL

BAFF

Sigma ③

100 $\times 10^9$ L

CD40 30 $\times 10^9$ L

2 30 $\times 10^9$ L

1 2 2

1.1

2017 12 2019 3

ITP 80

100

44 36 23-58 42.23 \pm 6.11

BMI 21-29 kg/m² 26.58 \pm 3.05 kg/m²

m² 55 45 21-59

42.22 \pm 6.31 Body Mass Index

dex BMI 20-28 kg/m² 26.33 \pm 2.88 kg/m²

①

5

②

③

① ITP ②

③

BMI $P > 0.05$ $P < 0.05$ 1

1.3

SPSS 22.0

$\bar{x} \pm s$ t

Perason

ROC

CD40 CD40L BAFF

ITP $P < 0.05$

2

2.1 2 CD40 CD40L

BAFF

CD40 CD40L

BAFF

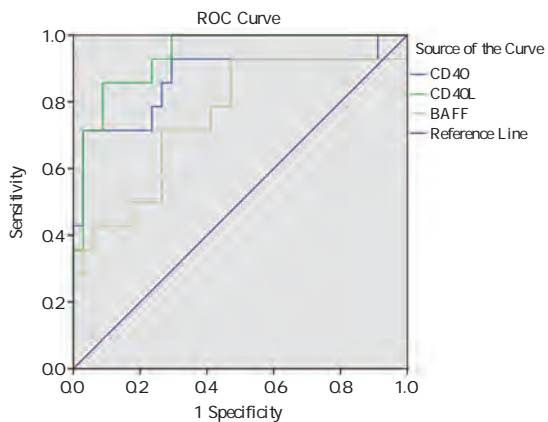
$P < 0.05$ 1

	1	2	CD40	CD40L	BAFF	2.2	CD40	CD40L
			$\bar{x} \pm s$					
Table 1 Comparison of CD40 CD40L expression and serum BAFF levels in peripheral blood of 2 groups of patients $\bar{x} \pm s$								
			CD40	CD40L	BAFF ng/L			
			7.31±2.07	3.24±1.06	603.17±112.38	2.3	ITP	
			12.92±3.05	6.09±1.70	716.44±137.48	BAFF		
t			6.721	5.947	2.936	ITP	CD40	CD40L
P			0.000	0.000	0.005		r	BAFF
						-0.713	-0.652	P<0.05

	2	CD40	CD40L	BAFF	$\bar{x} \pm s$
Table 2 Comparison of CD40 CD40L expression and serum BAFF levels in peripheral blood of patients with different curative effects in the observation groups $\bar{x} \pm s$					
	n	CD40	CD40L	BAFF ng/L	
	56	11.63±3.73	5.61±1.14	683.08±111.88	
	24	15.92±5.09	6.88±1.25	804.07±117.65	
t		2.127	2.237	2.155	
P		0.045	0.034	0.040	

2.4 ROC

ROC	CD40	ITP	3
cutoff	12.28 %	AUC 0.892	30% ITP
83.16%	92.06%	CD40L	
ITP	cutoff	6.08% AUC 0.784	6.7
	81.75%	90.25% BAFF ITP	ITP
	cutoff	745.02 ng/L AUC	T B
0.708	81.02%	90.14%	ITP ITP
1			



1 ROC
Figure 1 ROC analysis chart

8 13 ITP CD40
CD40L CD40L

T CD40 5

CD40L

ITP B ITP

B BAFF 9

ITP

BAFF ITP

14 15

ITP CD40 CD40L

BAFF

CD40 CD40L BAFF

ITP

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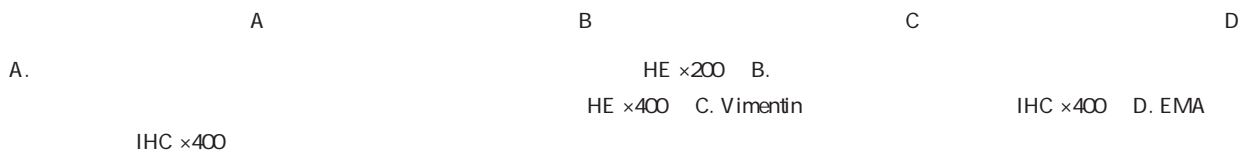
1

1 1 1 2 3 1★

1 SPRCC SPRCC 1 HE EnVision
SPRCC

1. 272111
2. 272111
3. 256610
* E-mail 461940072@qq.com

papillary renal cell carcinoma
 2016 12 5
 ma PRCC
 50% 3.7×3.6×2.1 cm
 solid variant of papillary renal cell carcinoma
 SPRCC
 PRCC 1 2.2 4×3×2 cm
 1
 10% HE
 CKpan CK7 CK8/18
 CK19 P504S CD10 EMA PAX 8 Vimentin
 RCC WT 1 CK20 CD57 Syn CD56 CgA WHO/ISUP 1 2
 CD117 Desmin SMA Myogenin S 100 E cadherin Ki 67 1A B 1C D 1
 2 2.3 39
 2.1 73 CT



1 SPRCC
 Figure 1 Histological characteristics and immunophenotype of SPRCC

1
 Table 1 The clinical datas and immunohistochemical results of SPRCC

73	4×3×2 cm	CKpan CK7 CK8/18 CK19 P504S CD10 EMA PAX 8 Vimentin Ki 67	RCC WT 1 CK20 CD57 Syn CD56 CgA CD117 Desmin SMA Myogenin S 100 E cadherin
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3

3.1

3.2.1 SPRCC

2

metanephric adenoma

5

80%~90%
PRCC

11

15cm

2

PRCC

I

II

I

3

II

I

12

II

4

PRCC

Vimentin
WT 1 EMA P504S

CK
CKpan CD57

12 13

50%

5

SPRCC

PRCC

Cadherin17

CDH17 81%

SPRCC

14

3.2.2 SPRCC

renal oncocytoma

6

2016

60-70

WHO

WHO/SUP

4

SPRCC

WHO/SUP1 2

7

CK 8/

18 CK19 EMA CK7 CD10 P504S PAX 8

Vimentin CK20 RCC WT 1 CD57

S 100 CD117 8 9

SPRCC

39

SPRCC

10

3.2

SPRCC

SPRCC

15

COPD
IL 17 ACTA

CysC VEGF

1 1 2 3*

A ACTA 2019 3 n=146

3 224

ACTA APACHE II P<Q05 VEGF CysC IL 17 ACTA P<Q05 VEGF APACHE II CysC VEGF IL 17 ACTA P<Q05 CysC VEGF IL 17 ACTA AUC 0.864 ACTA COPD COPD

C CysC COPD 86

VEGF 17 IL 17 2016 n=78 CysC VEGF IL 17

3 CysC VEGF IL 17 ACTA CysC IL 17 ACTA APACHE II VEGF CysC IL 17 ACTA APACHE II COPD CysC VEGF IL 17

17 A

Changes of serum CysC VEGF IL-17 and ACTA levels in elderly patients with COPD and severe respiratory failure

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ABSTRACT Objective To investigate the

Key words

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tion group and analyzed the diagnostic value of serum indicators for elderly COPD with severe respiratory failure and the correlation between serum indicators and the correlation between serum indicators and APACHE II score and the prognostic value were analyzed. Results The levels of serum CysC VEGF IL 17 and ACTA in the three groups were statistically significant $P<0.05$ VEGF was negatively correlated with CysC IL 17 and ACTA levels and CysC IL 17 and ACTA levels were positively correlated $P<0.05$ the serum levels of CysC IL 17 ACTA and APACHE II score were higher in dead patients than in surviving patients and VEGF levels were lower than in surviving patients $P<0.05$ VEGF level was negatively correlated with APACHE II score and CysC IL 17 and ACTA levels were positively correlated with APACHE II score $P<0.05$ the combined serum levels of CysC VEGF IL 17 and ACTA in predicting the prognosis of COPD with severe respiratory failure in elderly patients 0.864 was greater than that of a single serum indicator with a sensitivity of 92.86% and a specificity of 70.00%. Conclusion Serum CysC VEGF IL 17 and ACTA levels are abnormally expressed in elderly patients with COPD and severe respiratory failure and are closely related to the disease and the combined detection is expected to be an effective method for diagnosis and prognosis of elderly COPD with severe respiratory failure.

KEY WORDS Cystatin C Vascular endothelial growth factor Interleukin 17 Activin A Chronic obstructive pulmonary disease Respiratory failure

Chronic obstructive pulmonary disease COPD
 Cystatin C CysC
 Vascular endothelial growth factor VEGF
 Interleukin 17 IL 17
 Activin A AC

TA
 CysC VEGF IL 17 ACTA
 IL 17 ACTA
 COPD

COPD
 COPD
 COPD
 COPD

2016 3 2019 3 224
 n=78 n=146
 86
 P>0.05 1

n % $\bar{x} \pm s$

Table 1 Comparison of general data between the 2 groups n % $\bar{x} \pm s$

	n=78	n=146	n=86	F/ χ^2	P
/	45/33	84/62	49/37	0.010	0.995
	65.97±2.15	66.19±1.74	65.80±1.83	1.218	0.297
COPD	9.89±1.22	10.02±1.13	9.72±1.30	1.693	0.186
kg/m ²	22.79±2.20	23.15±2.08	22.94±2.13	0.779	0.460
	20 25.64	37 25.34	21 24.42		
	13 16.67	24 16.44	14 16.28	0.019	1.000
	8 10.26	14 9.59	8 9.30		

① COPD ②
 <60mmHg >
 50mmHg ③ >60 ④ 1.2
 ① ① 3 CysC VEGF IL 17 ACTA
 ② 6 ③ ② CysC VEGF IL 17 ACTA
 ③
 ④ ⑤ II APACHE
 II APACHEII 71 ④
 1.2 CysC VEGF IL 17 ACTA APA
 1.2.1 CHEII ⑤ CysC VEGF IL 17
 3mL 12min 3000r/min ACTA COPD
 - 70
 Bio Bad 1.5
 CysC VEGF IL 17 ACTA SPSS 22.0
 $\bar{x} \pm s$ t n %
 50 L μ 37 50 L μ Pearson χ^2
 30min ROC P<0.05
 5
 50 L μ 37 30min 2
 5 37 10min
 50 L μ 15min 2.1 3 CysC VEGF IL 17 ACTA
 450nm OD 3 CysC VEGF IL 17 ACTA
 1.2.2 P<0.05 CysC IL 17
 ACTA > > VEGF
 < < 2

Table 2 Comparison of serum CysC VEGF IL 17 and Acta levels between the 2 groups $\bar{x} \pm s$

	n	CysC ng/mL	VEGF mg/L	IL 17 ng/L	ACTA ng/L
	78	151.23±30.24 ^a	323.43±54.12 ^b	65.14±18.52 ^b	45.69±8.24 ^b
	146	130.47±24.58 ^b	387.64±69.12 ^a	44.87±15.76 ^b	36.51±6.25 ^a
	86	102.41±20.37	451.63±67.29	18.31±12.84	25.74±6.13
F		79.021	79.267	182.984	178.781
P		0.001	0.001	0.001	0.001

^aP<0.05

^bP<0.05

2.2 APACHE II VEGF
 VEGF CysC IL 17 ACTA P<0.05 3
 CysC IL 17 ACTA 2.4 APACHEII
 IL 17 ACTA P<0.05 1 VEGF r=- 0.349 APACHE II
 2.3 APACHEII CysC r=0.836 IL 17 r=
 28 50 0.654 ACTA r=0.675 APACHE II
 CysC IL 17 ACTA P<0.05

VEGF mg/L

450	100
400	80
350	60
300	40
250	20
200	

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		VEGF	
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		12 13	
COPD		IL 17	5 Celli B Decramer M Wedzicha JA et al. An Official American Thoracic Society/European Respiratory Society Statement Research Questions in Chronic Obstructive Pulmonary Disease J . Am J Respir Crit Care Med 2015 191 7 e4 e27.
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ACTA		COPD	9 Zhang M Li Y Yang X et al. Serum Cystatin C as an Inflammatory Marker in Exacerbated and Convalescent COPD Patients J . Inflammat 2016 39 2 625 631.
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CysC VEGF IL 17 ACTA			10 Byers DE. Linking VEGF Deficiency and IL 33 Upregulation in Chronic Obstructive Pulmonary Disease J . Am J Respir Cell Mol Biol 2019 61 5 550 551.
COPD		CysC VEGF IL 17	
ACTA		COPD	11 v . VEGF ICAM 1 IL 13
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IL 17 ACTA		COPD	J . 2017 22 9 1563 1566.
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¹⁸ Bergamaschi A ¹⁹
14 3 3ζ
14 3 3ζ microRNA 451
miR 451 14 3 3ζ
SERMs
Liu ZR ²⁰ miR 451a
MCF 7 LCC 2
miR 451a 14 3 3ζ 14 3 3ζ
14 3 3ζ miR 451a
14 3 3ζ
14 3 3ζ

FOXM 1
²¹ 14 3 3ζ

2.3 14 3 3ζ

²² 14 3 3ζ
70% -75% 14 3 3ζ
45% ²³ 14 3 3ζ

B 1 CDC25B birc5
²⁴

14 3 3ζ
Li Y

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MYC¹⁵ MYCN SHH Group4 MB
 1.4 Group 4 Group3 TP53
 Group 4 MB SHH³¹
 35%⁷ 5 75%¹¹ MB
 Group 4
 /
 EOMES LIM GTR MB
 1α LMX1A²⁸ 35%~40% Thompson NTR GTR
 Group 4
 KDM6A α Group4 MB NTR
 SNCAIP MYCN³² GTR
 CDK6 Northcott PRDM6 GTR
 4
 PRDM6 SNCAIP 600kb SNCAIP
 Group 4²⁶ 2.3

ERBB4 SRC Group 4²⁹ MB
 17q Group 4 MB
 Group 4α Group 4y CDK6 MB
 8p 7q Group
 4β SNCAIP PRDM6³⁰
 2

MB
 WNT MB 100%
 24 Gy 18Gy
 12.8% MB
 33 XRT
 PRT XRT

/
 2.1 3 2.4^{34 35}
 MB
 3

1.5 cm² MB
 4 >90%
 75%~90% 50%~75% SHH PCT1
 <50% Group 4 11 MB G SMO SHH
 WNT 17 Group 4 MB MYC
 Group3 Group 4 MB MYC
 Group3 SHH³⁶ SMO SHH
 Robinson Vismo

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