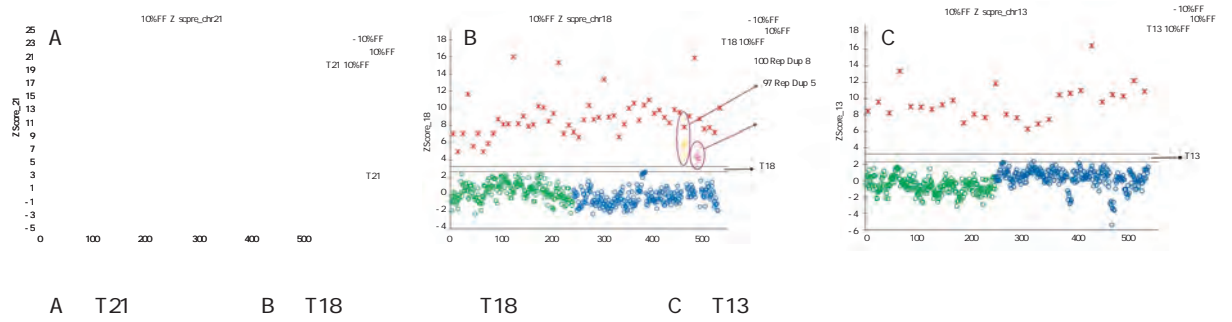


2020 11 12 11 75

Volume 12 Number 11 November 2020



P1436 10% cffDNA  
Figure P1436 The results of national reference materials with 10% cffDNA fraction

ISSN 1674-6929



1963 10

CLIN EXP INT OPHTHALMOL GRAEF ARCH  
THERAPY ADVANCES IN

360°

AMD PCV

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"

2017

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SCI

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Storz Alcon

# 分子诊断与治疗杂志

JOURNAL OF MOLECULAR DIAGNOSTICS AND THERAPY

2020 11 12 11 75 Bimonthly Volume 12 Number 11 November 2020

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# 分子诊断与治疗杂志

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# JOURNAL OF MOLECULAR DIAGNOSTICS AND THERAPY

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Progress of researches on Epstein Barr virus laboratory detection technology

# Angio OCT

Angio OCT

OCT Angio OCT

Angio OCT

Angio OCT

## Application progress of Angio OCT in comprehensive diagnosis and treatment of ocular fundus diseases

ZHONG Liting XIANG Wu ZHONG Yanfeng YI Ke ZHANG Shaochong

Zhongshan Ophthalmic Center Sun Yat sen University Guangzhou Guangdong China 510060

**ABSTRACT** Optical coherence tomography angiography Angio OCT is a new imaging technology used to detect blood flow in the fundus. Compared with traditional OCT Angio OCT has the advantages of high resolution safety speed and non invasiveness. Non invasive quantitative measurement of ocular anatomical structures was achieved such as retina choroidal blood vessels and blood perfusion. This article reviews the basic principles of Angio OCT quantitative measurement of blood flow transplantation and its application in fundus diseases such as choroidal neovascularization central serous chorioretinopathy diabetic retinopathy retinal arteriovenous and polypoid choroidal vascular diseases. The shortcomings are summarized to deepen the ophthalmology clinicians comprehensive knowledge of Angio OCT as a new inspection method.

**KEY WORDS** Optical coherence tomography angiography Polypoidal choroidal vasculopathy Central serous chorioretinopathy Diabetic retinopathy Retina artery and vein occlusion Polypoid choroidal vascular disease

tomography OCT

OCT

optical coherence

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OCT optical  
coherence tomography angiography Angio OCT

FFA ICGA Angio OCT

1 Angio OCT

Angio OCT ICGA  
Angio OCT

Angio OCT

ICGA SPAIDE<sup>15</sup> FFA Angio OCT  
FFA Angio OCT  
Angio OCT  
Angio OCT  
FFA ICGA

2

2 Angio OCT

3 Angio OCT  
split spectrum amplitude decorre-  
lation angiography SSADA  
Angio OCT

2.1 CNV  
wet age related macular degeneration wAMD  
OCT  
FFA ICGA CNV  
Angio OCT

4 3D  
X Y X Z Y Z

CNV  
17

Angio OCT

De CARLO<sup>18</sup> Angio OCT  
CNV  
Angio OCT  
CNV

5 6 7 8 9

in angiography FFA fundus fluoresce  
nine green angiography ICGA indocya

2.2 serous chorioretinopathy CSC Central

10 FFA ICGA 2D  
11 Angio OCT ICGA FFA

19

12

OCT CSC

Angio OCT

CSC

CSC

<sup>20</sup> Angio OCT

CSC

Angio OCT

CSC

ICGA  
 RPE  
 PCV  
 PCV  
 Angio OCT  
 Angio OCT  
 32  
 RPE  
 Angio OCT  
 PCV  
 pachyvessels  
 Angio OCT  
 PCV  
 Angio OCT  
 Angio OCT  
 Angio OCT  
 Angio OCT  
 An  
 Angio OCT

3 Angio OCT

Angio OCT

Angio OCT

OCT

6 μm

11 μm

ART=25

ART=7 4 7

ART=2

ART=100

Angio OCT

33

Angio OCT BRVO

BRVO

4 Angio OCT

Angio OCT

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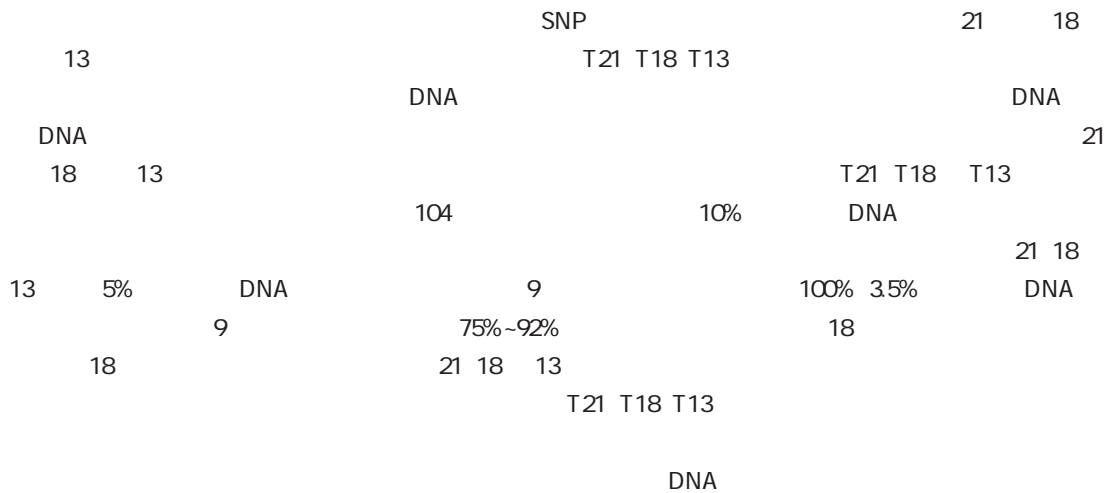
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## T21 T18 T13



Evaluation of fetal chromosome aneuploidy T21 T18 T13 21

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2016YFC1000300

100050

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tests were from 75% to 92%. The microduplication reference of chromosome 18 were all trisomy 18 while the results of other microdeletion and microduplication reference were not trisomy 21 18 and 13. The repeatability was in line with the requirement of national reference. Conclusion The national reference materials have good applicability and the performance of fetal chromosome aneuploidy detection kit probe hybridization meets its requirements

KEY WORDS Chromosomal aneuploidy single nucleotide polymorphism Cell free fetal DNA Trisomy T

21 18 13  
12 21 18 13

non invasive prenatal testing NIPT<sup>3 4</sup>  
single nucleotide  
polymorphism microarray SNP array  
<sup>5 6</sup> NIPT

T13 r##\*% % T18 # % # \*

density raw\_ra T21 T21 3  
tio Levenberg Marquardt  
Norm\_ratio Z  
21 18 13  
2  
2.1 T18  
3  
9 15  
10% DNA cell free fetal DNA cffDNA  
T21 32



	1	1	3	1	2	3	1	2	3	
	1	2	2	4	2	1	4	3	2	
	1	1	1	2	1	1	1	1	1	
	22	21	21	18	21	22	19	20	21	
%	92%	22/24	88%	21/24	88%	21/24	75%	18/24	88%	21/24

21 18 13 DNA  
 DNA  
 cfDNA  
 DNA  
 PCR  
 SNP  
 21 18 13  
 SNP  
 SNP DNA  
 DNA 10% 96%  
 DNA 4%<sup>9 10</sup> DNA  
 NIPT DNA  
 8  
 5% DNA  
 90% 3.5% DNA  
 50%<sup>9</sup>  
 104  
 9  
 5% DNA  
 100% 3.5% DNA  
 75% 92%  
 18

# ALK EGFR ROS1

NSCLC ALK EGFR ROS1  
2016  
1 2018 1 51 64 ALK EGFR  
ROS1 logistic  
NSCLC NSCLC ALK EGFR ROS1 EGFR  
P<0.05 ALK EGFR NSCLC  
ROS1 NSCLC EGFR NSCLC  
P<0.05 EGFR rs121434569 OR=56.00 ALK  
EGFR NSCLC EGFR NSCLC  
EGFR rs121434568  
ALK EGFR ROS1

## Polymorphisms of susceptibility genes *ALK*, *EGFR* and *ROS1* in non small cell lung cancer within Han population in Hubei province

SHEN Zhijun LIU Shiguo

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ABSTRACT Objective To study the mechanism of occurrence and development in non small cell Lung Carcinoma

10

5 25-30%  
Non small cell lung  
cancer NSCLC 85%<sup>1</sup>

SNP single nucleotide polymorphism  
<sup>2</sup> NSCLC

PCR NSCLC

1

1.1

2016 1 2018 1  
51 NSCLC  
36 70.6% 15 29.4% 62.4±  
14.1 X CT

NSCLC NSCLC

64  
35 54.7% 29  
45.3% 61.8±15.3

1.2

NSCLC logistic

P<0.05

2

2.1

NSCLC

NSCLC ALK EGFR

ROS1

EGFR P<

0.05 2

2.2 ALK EGFR ROS1

NSCLC

Logistic ALK rs113994090

rs113994092 NSCLC

P<0.05 EGFR rs28929495 rs121434568

rs121434569 rs12193428 rs121913465 rs397517127

rs606231253 NSCLC P<

0.05 ROS1 NSCLC

P>0.05 3

2.3 ALK EGFR ROS1

NSCLC

Logistic EGFR

NSCLC

rs121434569 ALK

NSCLC

P>0.05 4

3 ALK EGFR ROS1 logistic

Table 2 Comparison of clinical data in the two groups

n %		n %		t/ <sup>2</sup>	P
NSCLC		NSCLC			
n=64	n=51				
61.8±15.3	62.4±14.1			0.196	0.8045
35 54.7	36 70.6			3.038	0.087
29 45.3	15 29.4				
-	32 41.2				
28 43.8	9 17.6	8.862	0.003		
26 40.6	14 27.5	2.172	0.141		
10 15.6	28 54.9	19.790	<0.001		
276±1.65	259.89±345.51				
-	46 90.2				
-	3 5.9				
-	2 2.9				
1 1.6	6 11.8	5.123 <sup>a</sup>	0.024		
63 98.4	45 88.2				
3 4.7	31 60.8				
61 95.3	20 39.2	42.890	<0.001		
0 0	3 5.9				
64 100	48 94.1	3.866 <sup>a</sup>	0.049		

a fisher

Table 3 Univariate analysis of polymorphism of ALK EGFR and ROS1 by logistic regression

SNP	NSCLC	B	95%CI	r <sup>2</sup>	P
ALK					
rs113994090	0	4	0.577	0.083-1.070	0.037 0.023
rs113994092	0	4	0.577	0.083-1.070	0.037 0.023
rs281864719	0	1	0.561	-0.430-1.553	0.002 0.264
rs281864720	1	3	0.318	-0.184-0.819	0.005 0.213
EGFR					
rs28929495	0	11	0.615	0.322-0.909	0.125 <0.001
rs121434568	1	26	0.679	0.501-0.857	0.330 <0.001
rs121434569	1	24	0.660	0.472-0.848	0.294 <0.001
rs12193428	3	30	0.653	0.488-0.818	0.348 <0.001
rs121913465	1	18	0.604	0.381-0.826	0.197 <0.001
rs397517127	0	5	0.582	0.141-1.023	0.049 0.001
rs606231253	0	20	0.674	0.464-0.883	0.253 <0.001
ROS1	0	3	0.566	-0.134-1.267	0.014 0.112

4 ALK EGFR ROS1 logistic  
 Table 4 Binary Logistic Regression analysis of ALK  
 EGFR and ROS1 genes

SNP	OR	95%CI	P
ALK			
rs281864720	3.937	0.397-39.043	0.242
EGFR			
rs121434568	65.520	8.432-509.119	<0.001
rs121434569	56.000	7.205-435.243	<0.001
rs12193428	29.048	8.026-105.129	<0.001
rs121913465	34.364	4.392-268.874	0.001

ROS1 c ros NSCLC  
 ROS1

EGFR ALK 17 ROS1

NSCLC

EGFR /KRAS /ALK

ROS1

1.7% 18 ROS1

NSCLC

Single nucleotide polymorphism

SNP 1.5x10<sup>7</sup> SNP  
 300-600 bp 3 NSCLC

SNP D 4

TERT 3 4 TOP2A ERCC1 5 miR 423 3p  
 6 1 7 IL 17F

IL 17A 8 NSCLC

Genome wide association

GWAS <https://atlas.ctglab.nl/> 9

ALK EGFR ROS1

SNP NSCLC

ALK NSCLC 10 NSCLC ALK  
 1.4-13% 11 NSCLC  
 ALK NSCLC  
 12 ALK NSCLC

NSCLC

50% 30% 6

miRNA

LncRNA SNP  
 20 21

EGFR

90% EGFR NSCLC 18 21 13  
 Kosaka 14

30% 15 EGFR

NSCLC EGFR rs121434568  
 rs121434569 rs12193428 rs121913465 NSCLC  
 EGFR rs2293347

EGFR

1 J . 2020 28 2 330-334.  
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1.3

SPSS 23.0  
n %  
P<0.05  
2  
2.1  
4 983  
8.03% 14  
30 32 CD31  
14 338 107 231  
14 62 1  
2.2  
14 338 281  
80 201

1 400  
Table 1 Genotypic distribution of 400 patients with thalassemia

n	%
o/ N	
228	
CD41 42N	137 34.25
CD17N	56 14.00
CD71 72N	15 3.75
CD27 28N	11 2.75
CD43N	6 1.50
CD14 15N	1 0.25
IVS I 1N	1 0.25
IntN	1 0.25
+/ N	
161	
IVS II 654N	122 30.50
28N	35 8.75
29N	2 0.50
CAPN	1 0.25
IVS I 5N	1 0.25
E/ N	
11	
CD26N	11 2.75
	400 100

HbA 2

2 80  
Table 2 Comparison of red blood cell parameters between 80 male thalassemia carriers patients with different genotypes over 14 years old

	n	RBC ×10 <sup>12</sup> /L	HGB g/L	HCT %	MCV fl	MCH pg	MCHC g/L
o/ N	40	6.48±0.89	128±15.58	41.39±4.79	64.173±4.25	19.89±1.34	309.95±8.99
+/ N	36	6.44±0.65	130.58±12.35	42.08±4.15	65.46±5.16	20.32±1.74	310.56±8.68
E/ N	4	5.44±0.43	138.25±7.97	41.98±1.58	77.32±3.18	25.45±1.05	329.00±9.06
P <sup>a</sup>	-	0.840	0.494	0.507	0.237	0.223	0.767
F <sup>b</sup>	-	3.303	1.006	0.236	14.534	24.238	8.579
P <sup>b</sup>	-	0.042	0.371	0.790	0.000 <sup>c</sup>	0.000 <sup>c</sup>	0.000 <sup>c</sup>
	n	RDW %	HbA %	HbA 2 %	HbF %	E %	
o/ N	40	17.42±1.40	93.63±1.45	5.65±0.72	0.72±1.02	0	30.27±6.68
+/ N	36	15.99±1.34	93.85±1.46	5.26±0.72	0.89±1.33	0	30.31±6.15
E/ N	4	13.50±0.68	69.97±1.36	3.73±0.15	0.33±0.57	25.97±1.27	29.25±4.50
P <sup>a</sup>	-	0.000 <sup>c</sup>	0.557	0.027 <sup>c</sup>	0.667	-	0.982
F <sup>b</sup>	-	21.584	505.193	17.616	0.328	-	0.051
P <sup>b</sup>	-	0.000 <sup>c</sup>	0.000 <sup>c</sup>	0.000 <sup>c</sup>	0.772	-	0.950

a o/ N vs +/ N b o/ N vs +/ N vs E/ N c P<0.05

3

3 9 11  
5 34  
6 CD41 42 IVS II 654  
0 CD17 28 CD71 72 CD26<sup>12</sup>  
14  
67 3  
8 13 14 15 2

3 201 14 -  
 Table 3 Comparison of red blood cell parameters between 201 female thalassemia carriers patients with different genotypes over 14 years old -

	n	RBC $\times 10^{12}/L$	HGB g/L	HCT %	MCV fL	MCH pg	MCHC g/L
$\alpha/\alpha$	112	4.84 $\pm$ 0.73	98.24 $\pm$ 11.94	31.66 $\pm$ 4.02	65.70 $\pm$ 4.53	20.41 $\pm$ 1.56	310.61 $\pm$ 8.35
$\alpha^+/ \alpha$	82	4.77 $\pm$ 0.74	100.79 $\pm$ 13.36	32.07 $\pm$ 4.21	67.57 $\pm$ 5.70	21.25 $\pm$ 2.06	314.48 $\pm$ 10.89
$\alpha/\alpha^+$	7	4.50 $\pm$ 0.44	115.86 $\pm$ 14.63	35.21 $\pm$ 4.25	78.10 $\pm$ 3.04	25.71 $\pm$ 1.60	329.286 $\pm$ 10.87
P <sup>a</sup>	-	0.539	0.164	0.495	0.012 <sup>c</sup>	0.001 <sup>c</sup>	0.006 <sup>c</sup>
F <sup>b</sup>	-	0.832	6.759	2.525	21.455	31.392	14.643
P <sup>b</sup>	-	0.437	0.001 <sup>c</sup>	0.083	0.000 <sup>c</sup>	0.000 <sup>c</sup>	0.000 <sup>c</sup>

	n	RDW %	HbA %	HbA2 %	HbF %	E %	
$\alpha/\alpha$	112	16.27 $\pm$ 1.65	92.66 $\pm$ 3.22	5.40 $\pm$ 0.60	1.57 $\pm$ 1.92	0	27.64 $\pm$ 4.56
$\alpha^+/ \alpha$	82	15.337 $\pm$ 1.13	93.46 $\pm$ 1.46	5.17 $\pm$ 0.50	1.34 $\pm$ 1.33	25	28.03 $\pm$ 6.17
$\alpha/\alpha^+$	7	14.81 $\pm$ 2.43	69.42 $\pm$ 1.22	3.74 $\pm$ 0.36	0.33 $\pm$ 0.65	26.58 $\pm$ 0.76	28.29 $\pm$ 4.39
P <sup>a</sup>	-	0.000 <sup>c</sup>	0.120	0.035 <sup>c</sup>	0.475	-	0.583
F <sup>b</sup>	-	10.860	185.125	21.289	1.156	3.637	0.181
P <sup>b</sup>	-	0.000 <sup>c</sup>	0.000 <sup>c</sup>	0.000 <sup>c</sup>	0.318	0.129	0.834

a  $\alpha/\alpha$  vs  $\alpha^+/ \alpha$  b  $\alpha/\alpha$  vs  $\alpha^+/ \alpha$  vs  $\alpha/\alpha^+$  c P<0.05

16 97.50 fL 31.40 pg Int MCV MCH  
 17 83.30 fL 27.70 pg  
 18 CD41 42N IVS II 654N  
 19 CD41 42N IVS II 654N DNA  
 20 CD41 42 IVS II 654  
 2015 CD41 42 CD17 28 21 CD41 42N IVS II 654N  
 CD41 42N IVS II 654N 28N CD17N

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22 2013 4  
 2015 3 CD41 42 IVS II 654 CD17  
 CD41 42N CD17N

MCV MCH  
 MCV MCH  
 HbA2 23

2  $\alpha/\alpha$   
 $\alpha^+/ \alpha$  RBC MCV MCH  
 RDW CV HbA2

1 CAP MCV MCH

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J . 2018 36 5 21

787 789. J . 2013 13 11 1409

14 . 5042 1411+1428

J . 2011 16 22

3 125 128. J . 2015 8 8 50 52

15 . 941 . MCV MCH HbA2

J . 2017 34 2 J . 2017 24

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1442

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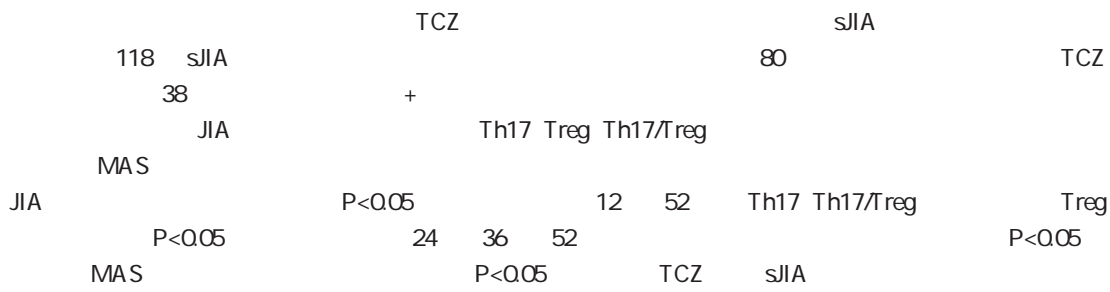
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TCZ

sJIA

Th17/Treg



### Effect of TCZ treatment on peripheral blood Th17/Treg and inflammatory indexes in children with sJIA

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**ABSTRACT** Objective To explore the effect of tocilizumab TCZ treatment on children with systemic juvenile idiopathic arthritis sJIA . Methods 118 children with sJIA in our hospital were selected retrospectively as the research objects. According to the treatment plan the observation group 80 cases was treated with glucocorticoid combined with TCZ and the control group 38 cases was treated with glucocorticoid + methotrexate and/or leflunomide. The main efficacy indicators secondary efficacy indicators during treatment JIA core evaluation parameters peripheral blood Th17 Treg Th17/Treg discontinuation of glucocorticoids macrophage activation syndrome MAS severe infection severe liver damage and the incidence of drug withdrawal were compared between the two groups. Results The main efficacy indicators secondary efficacy indicators and JIA core evaluation parameters in the observation group were better than those in the control group P<0.05 Th17 and Th17/Treg of the observation group were lower than those of the control group after 12 and 52 weeks of treatment and Treg was higher than that of the control group P<0.05 the proportion of discontinuation of glucocorticoids after 24 weeks 36 weeks and 52 weeks in the observation group was higher than that in the control group P<0.05 . The incidence of MAS and severe infection in the observation group was lower than that in the control group P<0.05 . Conclusion TCZ has a significant effect in treating sJIA which can effectively reduce the inflammation in children regulate the immune function of the body reduce the use of glucocorticoid and has high safety.

**KEY WORDS** Tocilizumab Glucocorticoid Methotrexate Leflunomide Systemic juvenile idiopathic arthritis

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Juvenile Idiopathic Arthri

tis JIA Becton Dickinson FACScan Th17  
 sJIA JIA systemic JIA Th17/Treg  
 JIA 2/3 JIA 10% 1.3 12 52  
 sJIA IL 6 2 sJIA IL 6 3  
 sJIA IL 6 2 sJIA IL 6 3  
 TCZ IL 6 2 sJIA IL 6 3  
 Th/Treg sJIA Th17/Treg 3  
 sJIA TCZ 4  
 1.1 118 sJIA 80 38  
 P>0.05 sJIA 4  
 1.2 2 1 TCZ 30kg 12mg/kg  
 8mg/kg <30kg 100mL  
 0.9 >1h 12 4  
 1.2.2 3mL 52  
 ESR CRP P<0.05 2

CHAO 6  
 ACRPedi30 ACRPedi50  
 ACRPedi70 ACRPedi90  
 Th17 Treg Th17/Treg MAS  
 SPSS 22.0  
 n %  
 t P<0.05  
 P>0.05  
 n %

Table 1 Comparison of general data between 2 groups  
 n %

	80	38	<sup>2</sup> t	P
	50 62.50	24 63.16	0.005	0.945
	30 37.50	14 36.84		
	7.32±2.40	7.61±2.26	0.625	0.533
Hb	57 71.25	27 71.05	0.001	0.982
ESR	75 93.75	35 92.11	0.004	0.952
	53 66.25	26 68.42	0.055	0.815
	58 72.50	29 76.32	0.194	0.660
	80 100.00	38 100.00		
	56 70.00	27 71.05	0.014	0.907
	24 30.00	11 28.95		

Hb ESR  
 2.2  
 12  
 52  
 P<0.05 2

2  
Table 2 Comparison of disease inactivity ratio and clinical remission rate between 2 groups n %

	n	12	52
	77	34 44.16	65 84.42
	37	8 21.62	23 62.16
<sup>2</sup>		5.453	7.030
P		0.020	0.008

2.3

P<0.05 3

JIA

12 52

2.4

ESR CRP

CHAQ

P<0.05

1

3

4

3  
Table 3 Comparison of 2 groups of secondary efficacy indicators n %

	n	ACRPedi 30	ACRPedi 50	ACRPedi 70	ACRPedi 90
12	77	48 62.34	23 29.87	10 12.99	0 0.00
	37	13 35.14	6 10.81	2 5.41	0 0.00
<sup>2</sup>		7.434	5.023	0.827	
P		0.006	0.025	0.363	
52	77	77 100.00	75 97.40	72 93.71	63 81.82
	37	37 100.00	34 91.89	27 72.97	21 56.76
<sup>2</sup>			0.734	7.512	8.095
P			0.392	0.006	0.004

3

1

4 JIA

Table 4 Comparison of two groups of JIA core evaluation parameters

	n	cm	cm	ESR mm/h	CRP mg/L	CHAQ		
	77	7.48±2.05	6.91±1.46	6.76±1.30	6.37±1.27	78.43±22.71	10.77±3.02	51.10±8.46
	37	7.41±2.08	6.83±1.52	6.65±1.34	6.28±1.19	76.57±20.36	10.64±3.10	51.75±8.82
t		0.170	0.270	0.419	0.361	0.423	0.213	0.379
P		0.865	0.787	0.676	0.719	0.673	0.831	0.706
12	77	3.49±1.03	2.69±0.54	2.81±0.43	2.65±0.42	10.21±3.05	2.44±0.73	76.32±10.25
	37	5.08±1.27	3.47±0.61	3.39±0.55	3.13±0.51	16.85±4.11	4.03±0.82	63.49±9.14
t		7.143	6.920	6.144	5.322	9.687	10.457	6.474
P		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
52	77	1.33±0.40	1.01±0.29	1.13±0.23	1.07±0.30	4.15±1.06	0.62±0.20	108.43±9.09
	37	1.79±0.46	1.37±0.30	1.57±0.31	1.49±0.46	8.49±2.27	1.47±0.41	93.45±9.76
t		5.472	6.137	8.512	5.844	13.951	14.914	8.043
P		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

3

1

2.5 Th17/Treg

P<0.05

7

12 52 Th17 Th17/Treg

3

Treg

P<0.05

5

sJIA

2.6

24 36 52

5

P<0.05

6

sJIA

2.7

MAS



# VEGF Cydin D1 E Cadherin

1 2 3

RB VEGF

Cyclin D1 E Cadherin

2017 1 2019 1 55 RB SP

VEGF Cydin D1 E Cadherin VEGF Cydin D1 E Cadherin

RB VEGF Cydin D1

E Cadherin P<0.05

Spearman VEGF Cydin D1 E Cadherin P<0.05 RB

VEGF Cydin D1 E Cadherin P>0.05

RB VEGF Cydin D1 E Cadherin

P<0.05 Cox VEGF

Cyclin D1 E Cadherin P<0.05 VEGF Cydin D1

E Cadherin RB VEGF Cydin D1

E Cadherin RB

## Expression of VEGF Cydin D1 and E Cadherin in pediatric retinoblastoma and their relationship with histopathological characteristics

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**ABSTRACT** Objective To investigate the expression of vascular endothelial growth factor VEGF cell cycle regulatory protein Cydin D1 and epithelial cadherin E Cadherin in pediatric retinoblastoma RB tissues and their relationship with histopathological characteristics. Methods 55 tissue samples from pediatric RB patients and normal controls were collected in our Hospital from January 2017 to January 2019. Immunohistochemical SP method was used to detect and compare the expression of VEGF Cydin D1 and E Cadherin and analyze VEGF Cydin D1 and E Cadherin expression correlation and the relationship between the three indicators and histopathological characteristics of children. Results The results of

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immunohistochemical staining showed that the positive rates of VEGF and Cyclin D1 expression in the RB tissues were significantly higher than those in the normal retinal tissues and the expression of E Cadherin was significantly lower than that in the normal retinal tissues. The difference was statistically significant  $P < 0.05$ . Spearman correlation analysis showed that the expression of VEGF Cyclin D1 and E Cadherin were positively correlated  $P < 0.05$ . There was no statistically significant difference in the positive rates of VEGF Cyclin D1 and E Cadherin expression in children with RB in different genders ages eye categories  $P > 0.05$ . However there was significant difference in the positive rates of VEGF Cyclin D1 and E Cadherin expression in children with different differentiation clinical stage optic nerve invasion and pathological stage were compared with statistical significance  $P < 0.05$ . The results of Cox multivariate analysis showed that the higher the clinical stage the higher the degree of neurological invasion and the high expression of VEGF Cyclin D1 and the low expression of E Cadherin were the adverse factors affecting the survival of patients  $P < 0.05$ . Conclusion The higher the positive expression of VEGF and Cyclin D1 and the lower the positive expression of E Cadherin the higher the malignant degree of RB tumors in children and the worse the prognosis. The detection of VEGF Cyclin D1 and E Cadherin expression is helpful for the assessment of the condition of children with RB.

KEY WORDS Children Retinoblastoma Vascular endothelial growth factor Cell cycle regulatory protein Epithelial cadherin

retinoblastoma RB

1

vascular endotheli

al growth factor VEGF

2

D1 Cell cycle regulatory protein

D1 Cyclin D1

G1/S

3

Epithelial cadherin E Cadherin

4

VEGF Cyclin D1 E Cadherin RB

RB

VEGF

Cyclin D1 E Cadherin

RB

RB VEGF Cyclin D1 E Cadherin

1

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SPSS 20.0

n %

<sup>2</sup>

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RB

VEGF Cydin D1

E Cadherin

RB

VEGF Cydin

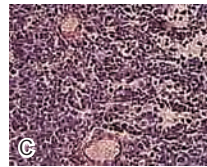
D1

E Cadherin

P<0.05

1

1



A

B

C

A B C VEGF Cydin D1 E Cadherin

1 RB VEGF Cydin D1 E Cadherin

SP ×200

Figure 1 Immunohistochemical staining of VEGF Cydin D1 and E Cadherin expression in RB tissue SP ×200

1

RB

VEGF Cydin D1

E Cadherin

n %

Table 1 Comparison of VEGF Cydin D1 and E Cadherin expression between normal retinal tissue and RB tissue

n %

		RB	<sup>2</sup>	P
VEGF		31 56.4	7 12.7	36.881 0.000
	+	16 29.1	10 18.2	
	++	6 10.9	20 36.4	
	+++	2 3.6	18 32.7	
Cydin D1		12 21.8	4 7.2	48.418 0.000
	+	37 67.3	9 16.4	
	++	5 9.1	27 49.1	
	+++	1 1.8	15 27.3	
E Cadherin		0 0.0	10 18.2	11.000 0.001
	+	4 7.3	26 47.3	
	++	9 16.4	8 14.5	
	+++	42 76.4	11 20.0	

2.2

RB VEGF Cydin D1 E Cadherin

VEGF Cydin D1

P<0.05 VEGF Cydin D1

E Cadherin

P<0.05

2

2 RB

VEGF Cydin D1 E Cadherin

Table 2 Correlation analysis of VEGF Cydin D1 and E Cadherin expression in RB tissue

	VEGF		Cydin D1		E Cadherin	
	r	P	r	P	r	P
VEGF	-	-	0.524	0.000	-0.493	0.000
Cydin D1	0.524	0.000	-	-	-0.508	0.000
E Cadherin	-0.493	0.000	-0.508	0.000	-	-

2.3

RB VEGF Cydin D1 E Cadherin

RB

VEGF Cydin

D1 E Cadherin

P>0.05

RB

VEGF Cydin D1

E Cadherin

P<0.05

3

2.4 Cox

VEGF

Cydin D1

E Cadherin

P<0.05

4

3

RB

6

7

8

herin RB

VEGF Cydin D1 E Cad

VEGF

9

Cydin D1

Cydin D1

3 RB VEGF Cydin D1 E Cadherin n %  
 Table 3 The relationship between the expression of VEGF Cydin D1 and E Cadherin and histopathological characteristics in children with RB n %

		VEGF		Cydin D1		E Cadherin	
		26 47.3	4 7.3	28 50.9	2 3.6	25 45.5	5 9.1
		22 40.0	3 9.1	23 41.8	2 3.6	20 36.4	5 9.1
<sup>2</sup>		0.022		0.036		0.102	
P		0.883		0.850		0.750	
	<3	29 52.7	5 9.1	32 58.2	2 3.6	28 50.9	6 10.9
	3	19 34.5	2 3.6	21 38.2	0 0.0	17 30.9	4 7.3
<sup>2</sup>		0.314		1.282		0.017	
P		0.575		0.258		0.896	
		26 47.3	3 5.5	26 47.3	3 5.5	23 41.8	6 10.9
		22 40.0	4 7.3	25 45.5	1 1.8	22 40.0	4 7.3
<sup>2</sup>		0.313		0.859		0.259	
P		0.576		0.354		0.611	
		35 63.6	0 0.0	35 63.6	0 0.0	11 20.0	9 16.4
		13 23.6	7 14.6	16 29.1	4 7.3	34 61.8	1 1.8
<sup>2</sup>		14.036		7.549		15.195	
P		0.000		0.000		0.000	
		2 3.6	5 9.1	3 5.5	4 7.3	24 43.6	4 7.3
		26 47.3	2 3.6	28 50.9	0 0.0	19 34.5	1 1.8
		20 36.4	0 0.0	20 36.4	0 0.0	2 3.6	5 9.1
<sup>2</sup>		25.419		29.580		15.963	
P		0.000		0.000		0.000	
	R0	1 1.8	4 7.3	2 3.6	3 5.5	22 40.0	1 1.8
	R1	25 45.5	2 3.6	26 47.3	1 1.8	23 41.8	4 7.3
	R2	22 40.0	1 1.8	23 41.8	0 0.0	0 0.0	5 11.1
<sup>2</sup>		22.514		22.927		25.665	
P		0.000		0.000		0.000	
		36 65.5	0 0.0	36 65.5	0 0.0	12 21.8	7 12.7
		12 21.8	7 12.7	15 27.3	4 7.3	33 60.0	3 5.5
<sup>2</sup>		15.197		8.173		6.795	
P		0.000		0.004		0.009	

4 Cox

Table 4 Cox regression multivariate survival analysis

	S.E	Wald	95%CI	P	
	0.813	0.438	5.124	1.115-4.561	0.024
	0.921	0.214	18.522	1.651-3.821	0.000
VEGF	1.034	0.301	11.801	1.559-5.073	0.002
Cydin D1	0.933	0.384	5.903	1.198-5.396	0.016
E Cadherin	-0.813	0.327	6.181	0.234-0.842	0.013

E Cadherin

<sup>13 14</sup> E Cadherin

<sup>15</sup> E Cadherin

E Cadherin

Cydin D1

VEGF Cydin D1

VEGF Cydin D1 E Cadherin

Cydin D1 <sup>10</sup>

RB Cydin D1

RB

Cydin

D1

VEGF Cydin D1

E Cadherin

herin RB VEGF Cyclin 7  
 D1 E Cadherin RB J . 2017  
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RB 9  
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 2019 11 2 117 121 127.

11 Th17/Treg J . 2019 59 25 27 30.

## CML Asprosin 2

2 T2DM N CML Asprosin  
 CAS 135 T2DM  
 IMT CAS =79 NCAS =56 60  
 NC ELISA  
 CML Asprosin Pearson CML Asprosin  
 logistic CML Asprosin T2DM CAS  
 CML Asprosin NCAS NC NCAS NC 3 P<  
 0.05 CML Asprosin FPG HOMI IR TC TG LDL C  
 HbA1C P<0.05 FPG HOMI IR TC TG  
 CML Asprosin logistic CML Asprosin FPG HOMI IR  
 T2DM CML Asprosin  
 2 N

### Relationship between CML Asprosin and carotid atherosclerosis in type 2 diabetes

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**ABSTRACT** Objective To investigate the relationship between serum N carboxymethyllysine CML and Asprosin levels and carotid atherosclerosis CAS in patients with type 2 diabetes T2DM . Methods 135 patients with T2DM were divided into the carotid atherosclerosis group CAS group 79 and the non carotid atherosclerosis group NCAS group 56 according to carotid ultrasound examination of carotid intima media thickness IMT another 60 healthy people were selected as the control group NC group . The clinical general data and blood biochemical indicators of each group were compared and the serum CML and Asprosin were detected by enzyme linked immunosorbent assay ELISA . The correlation between CML and Asprosin and other indicators were analyzed by Pearson correlation analysis and multiple linear regression analysis. Logistic regression analysis was used to analyze whether CML and Asprosin were risk factors for carotid atherosclerosis progression. Results In the T2DM combined with CAS group CML and Asprosin were significantly higher than those in the T2DM without CAS group and the control group. And the non CAS group was significantly higher than in the control group. The differences among the three groups were statistically significant P<0.05 . Pearson correlation analysis results showed that there was a positive correlation between CML Asprosin and the duration of diabetes FPG HOMI IR TC TG LDL C P<0.05 . The multiple lin

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ear regression results showed that the course of diabetes FPG HOMI IR TC TG are independent risk factors that affect the levels of CML and Asprosin. Logistic regression analysis showed that CML Asprosin and the course of diabetes FPG and HOMI IR were all risk factors for carotid atherosclerosis in patients with type 2 diabetes. Conclusion CML and Asprosin are significantly increased in patients with T2DM and carotid atherosclerosis suggesting that glycolipid toxicity and oxidative stress lead to endothelial damage and promote the occurrence and progression of carotid atherosclerosis.

KEY WORDS Type 2 diabetes Carotid atherosclerosis N carboxymethyllysine Asprosin

2 Type 2 diabetes mellitus T2DM BMI  
P>0.05  
1.2  
T2DM 1.2.1  
1 T2DM SONOS 5500  
7-11 MHz  
carotid atherosclerotic sclerosis CAS T2DM 1 cm 4  
2 N 1.2.2  
N carboxymethyl lysine CML 5 mL 3 000 r/  
min 15 min - 80  
3 CML 7600  
4 Asprosin Total cholesterol TC Tri  
T2DM 5 glyceride TG High density  
lipoprotein cholesterol HDL C  
T2DM CML Asprosin Low density lipoprotein cholesterol LDL C  
CML Asprosin T2DM CAS Glycated hemoglobin HbA1C  
1 Fasting plasma glucose FPG  
1.1 ELISA CML Asprosin  
2018 1 2019 1  
135 T2DM  
IMT 1.3  
CAS IMT 1.0 mm 1 SPSS 19.0  
6 CAS n=79 CAS n=56 n % 2 -  
60 t  
LSD t  
7 WHO T2DM Pearson CML  
18 Asprosin logistic  
1 P<0.05  
2  
2.1 3  
3  
3 BMI P>0.05 T2DM

CAS DBP SBP FPG TC TG LDL C HbA1c P<0.05 T2DM CAS  
 HOMA IR CML Asprosin NCAS NCAS  
 NC HDL C NCAS NC P<0.05 1

/

- %
- %
- BMI kg/m<sup>2</sup>
- DBP mmHg
- SBP mmHg
- FPG mmol/L
- HbA1c %
- HOMA IR
- TC mmol/L
- TG mmol/L
- LDL C mmol/L
- HDL C mmol/L
- CML µg/L

0

H 9 d2 m4 p ð Á1aÐ „1 m5 „&Ð P 2M  
 2.3 T2DM CML Asprosin @ ð € Á Ð # m5 P Á ð „2M  
 ð € Á Ð # m5 P Á ð „2M  
 P ( UZ 5 10 hC\$Ð

FPG HOMI IR TC TG

9ÖC Nü# Ú`À Á1Ð \$cE` ðNü# CML# Asprosin Ô !0 ðNü# q?!0 ðNü# CML# Asprosin Ü Nü# ÑÀ ðNü# 9G ðNü# 4d0 ðNü# ÖC Nü#  
 FPG HOMI IR TC TG LDL C HbA1C 2.4 T2DM  
 P<0.05 2 logistic

2 CML Asprosin

Table 2 Correlation analysis of CML Asprosin and clinical indicators in Patients

	CML		Asprosin	
	r	P	r	P
CML	-	-	0.501	0.016
Asprosin	0.501	0.016	-	-
FPG	0.551	0.008	0.582	0.017
HOMI IR	0.467	0.009	0.470	0.010
TC	0.709	0.004	0.740	0.000
TG	0.514	0.025	0.533	0.019
LDL C	0.486	0.021	0.546	0.001
HDL C	0.467	0.036	0.486	0.007
HbA1c	-0.496	0.097	-0.512	0.081
	0.538	0.026	0.603	0.019

CML Asprosin  
 TG LDL C

FPG HOMI IR TC  
 logistic

CML Asprosin

FPG HOMI IR  
 4

3

T2DM

T2DM

8

advanced

glycosylation end products AGEs

AGEs

NF kB

2.3 T2DM CML Asprosin

CML Asprosin

FPG HOMI IR TC TG LDL C HbA1C

Table 3		Multiple	3 T2DM	CML	Asprosin	P	Q	n	Q	S	U
				P	S.E.	OR	95%CI				P
CML				2.108	0.069	2.961	2.930-2.991				0.003
		FPG		0.737	0.209	2.074	1.380-3.116				0.0160
		HOMI IR		1.138	0.3629	1.149	1.042-1.506				0.000
		TC		1.632	0.736	5.136	1.217-21.596				0.022
Asprosin		TG		0.971	0.308	1.350	0.552-1.809				0.000
		FPG		1.906	0.675	6.736	0.716-1.406				0.003
		HOMI IR		1.034	0.301	1.352	0.552-1.816				0.016
		TC		2.186	0.587	8.862	0.315-3.138				0.010
		TG		0.874	0.273	1.321	0.573-1.726				0.031
				1.850	0.576	6.406	2.083-19.719				0.001

# D

D D D D TEG AICH 56  
 116 AICH  
 D D TEG AICH  
 D D R K > > MA CI  
 < < P<0.05 AICH  
 D D R K MA CI P<0.05 D D  
 R K MA CI P<0.05 AICH  
 D D R K MA CI P<  
 0.05 D D TEG K MA AICH AUC 0.899  
 AUC 95.24% 83.16% D D TEG  
 AICH  
 D

## Evaluation value of D dimer combined with thrombus elasticity chart on the condition and treatment outcome of patients with acute cerebral hemorrhage

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**ABSTRACT** Objective To explore the value of D dimer D D combined with thrombus elasticity map TEG in evaluating the condition and treatment outcome of patients with acute intracerebral hemorrhage AICH . Methods 116 cases of AICH patients in our hospital were selected as the study group and 56 healthy medical examiners in the same period were randomly selected as the control group. The serum D D and TEG parameters of the two groups were compared and the value of the above indicators for the assessment of AICH condition and the prediction of treatment outcome was analyzed. Results The results of comparison of serum D D R value and K value large bleeding patients in the study group>small bleeding patients>the control group and the result of comparison of angle MA value and CI value a large number of bleeding patients in the study group<a small amount of bleeding patients<the control group the difference was statistically significant P<0.05 . The severity of AICH patients was significantly correlated with serum D D R value K value angle MA value CI value the difference was statistically significant P<0.05 . The serum D D R and K values of the deceased patients in the study group were higher than those in the surviving patients and the angle MA value and CI value were lower than those in the surviving patients the difference was statistically significant P<0.05 . There was a positive correlation between serum D D and R value and K value in AICH

patients and a negative correlation between angle MA value and CI value the difference was statistically significant  $P < 0.05$ . The area under the curve AUC of the combined prediction of the K value and MA value of the serum D D and TEG parameters for the treatment outcome of AICH patients was 0.899 which was greater than the single predicted AUC of each indicator. The best sensitivity of the combined prediction was 95.24% and the specificity was 83.16%. Conclusion Serum D D and TEG parameters are closely related to the status of disease in patients with AICH and the combination of the two has a high application in the prediction of patient treatment outcome

KEY WORDS Acute cerebral hemorrhage D dimer Thromboelastography Bleeding volume Treatment outcome

1.2  
 20%~30%  
 Acute intracerebral hemorrhage  
 AICH 30%~40%  
 12 AICH  
 3  
 D D  
 D Dimer D D  
 UniCel DxC800 Synchron  
 TEG  
 GE 5000 TEG  
 3 mL TEG  
 R  
 K  
 MA CI  
 D D  
 1.3  
 D D TEG  
 R K MA CI AICH D D  
 1  
 1.1  
 2016 10 2019 10 116  
 AICH 74 42  
 51~75 45~88 kg CT  
 30 mL 42  
 <30 mL 74 6  
 56  
 36 20 48~73 47~85 kg  
 AICH  
 7  
 1.4  
 SPSS 22.0  
 - t  
 n %  
 Logistic Pearson  
 ROC  
 P<0.05  
 2  
 2.1  
 P>0.05 1

Table 1 Comparison of General information of 2 groups

	n=116	n=56	t/ <sup>2</sup>	P
/	42/74	20/36	0.004	0.950
kg	62.34±5.66	61.49±5.75	0.918	0.360
	65.39±10.19	63.51±8.25	1.203	0.231
	18 15.52	7 12.50	0.277	0.599
	25 21.55	10 17.86	0.318	0.573
	11 9.48	3 5.36	0.397	0.529
	68 58.62	30 53.57	0.393	0.531
	48 41.38	26 46.43		
	75 64.66	34 60.71	0.253	0.615
	41 35.34	22 39.29		

2.2 D D TEG  
 D D R K  
 MA CI  
 P<0.05 2

2.3 D D TEG  
 AICH  
 K MA CI  
 D D R  
 Logis  
 MA

Table 3 Relationship between serum D D and TEG parameters and the severity of AICH

	S.E.	Wald/ <sup>2</sup>	OR	95%CI	P
D D	1.774	0.528	11.286	5.893	4.279-8.116 <0.001
R	1.658	0.436	14.460	5.248	2.754-10.002 <0.001
K	1.570	0.511	9.443	4.808	3.158-7.320 <0.001
MA	-0.614	0.209	8.634	0.541	0.320-0.915 <0.001
CI	-0.786	0.234	11.284	0.456	0.237-0.876 <0.001
CI	-0.507	0.171	8.779	0.602	0.402-0.903 <0.001

2.4 D D TEG  
 1  
 D D R K MA  
 CI P<0.05 4  
 2.5 Pearson AICH D D R  
 K MA CI  
 r=0.645 0.620 - 0.481 - 0.554 - 0.629 P<0.05  
 1  
 2.6 D D TEG

Table 2 Comparison of serum D D and TEG parameters

	n	D D µg/L	R min	K min	deg	MA mm	CI
	42	344.49±82.25	9.77±1.02	4.51±0.57	40.53±5.04	47.54±4.06	1.26±0.18
	74	246.77±70.39	8.75±0.93	3.24±0.48	48.18±6.30	55.11±4.47	1.45±0.22
	56	130.42±31.15	7.07±1.38	2.30±0.41	60.04±10.20	59.26±7.13	1.63±0.37
F		137.331	74.848	251.492	84.416	57.049	22.538
P		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

Table 4 Comparison of serum D D and TEG parameters between surviving and dead patients

	n	D D µg/L	R min	K min	deg	MA mm	CI
	95	211.49±63.35	8.53±1.12	3.28±0.46	46.51±5.39	54.44±6.31	1.43±0.25
	21	601.80±187.44	11.79±1.89	5.60±0.75	40.43±4.06	43.01±5.22	1.15±0.14
t		16.631	10.490	18.409	4.866	7.729	4.953
P		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

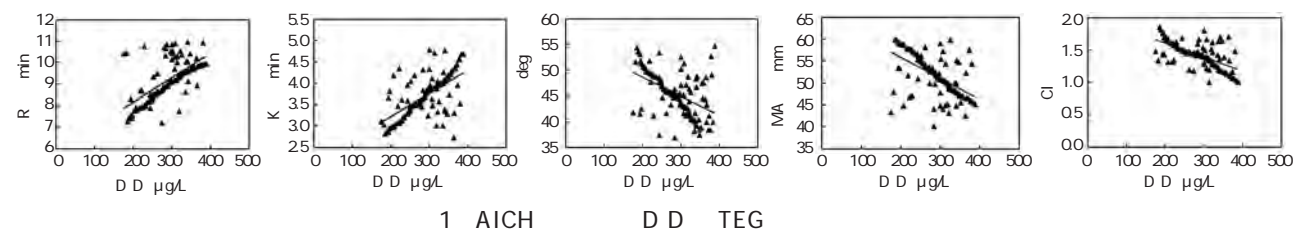


Figure 1 Relationship between serum D D and TEG parameters in AICH patients

ROC  
Area under the curve  
AUC 0.823 5 2  
2.7  
D D TEG  
ROC  
AUC  
0.899 95%CI 0.830-0.947 Z =13.214  
95.24% 83.16%

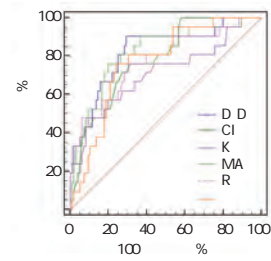


Figure 2 ROC of treatment outcome of AICH patients with single prediction of serum D D and TEG parameters

5 D D TEG AICH

Table 5 The value of serum D D and TEG parameters to predict the treatment outcome of AICH patients

	AUC	95%CI	Z		%	%	P
D D	0.823	0.741-0.888	6.080	>375.99µg/L	90.48	70.53	<0.001
R	0.772	0.685-0.845	0.772	>9.79 min	76.19	69.47	<0.001
K	0.708	0.617-0.789	2.927	>4.80 min	47.62	90.53	<0.001
	0.768	0.680-0.841	5.186	42.20 deg	76.19	75.79	<0.001
MA	0.819	0.736-0.884	5.967	49.64 mm	76.19	80.00	<0.001
CI	0.780	0.694-0.852	5.473	1.29	76.19	68.42	<0.001

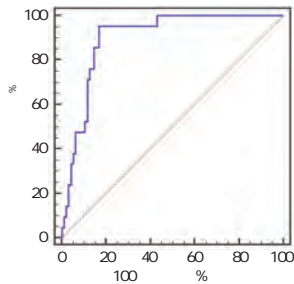


Figure 3 The combination of serum D D and TEG parameters to predict the ROC of treatment outcome in patients with AICH

ROC  
AUC 0.823  
375.99µg/L  
70.53%  
90.48%  
AICH TEG R K  
MA CI TEG

3

AICH D D  
8  
D D AICH  
AICH  
D D  
D D

Cheng 9

14

TEG  
TEG  
TEG AICH  
TEG AICH

12 TEG

13

R K  
MA CI

TEG AICH D D 7  
 D D  
 TEG J .  
 ROC D D TEG K 8  
 MA AICH AUC  
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 2015 24 12 1319 1323  
 D  
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 2018 31 3 219 221.  
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1460  
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 promotes foam cell apoptosis and atherosclerosis progression  
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 centrations are increased in type 2 diabetes mellitus and inde  
 pendently associated with fasting glucose and triglyceride J .  
 Clin chim acta intern J clin chem 2019 489 1 183 188  
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 N  
 J .  
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 N  
 2018 43 12 1613 1618.

# miR 34a

RNA miR 34a

2017 5 2019 5 142

86 miR 34a bcl 2 CCND1 Notch1

miR 34a pCR miR 34a pCR ROC

$t=4.321 P<0.05$  T N Ki 67

miR 34a P<0.05 miR 34a

bcl 2 CCND1 Notch1 miR 34a < P<0.05

0.05 ROC miR 34a pCR  $t=3.660 P<$

miR 34a miR 34a pCR

miR 34a

## The clinical significance of the change of miR 34a expression in peripheral blood of breast cancer patients

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**ABSTRACT** Objective To study the change of miR 34a expression in peripheral blood of breast cancer patients and its clinical significance. Methods 142 cases of breast cancer patients who received neoadjuvant chemotherapy in our hospital from May 2017 to May 2019 were selected as the breast cancer group and 86 cases of healthy people in the same period were selected as the control group. The expression level of miR 34a in peripheral blood of two groups and the expression of bcl 2 CCND1 Notch1 in breast cancer lesion were detected. Pathological complete response pCR of breast cancer patients were evaluated after neoadjuvant chemotherapy and surgery. ROC curve was used to analyze the predictive value of miR 34a for pCR. Results The expression level of miR 34a in peripheral blood of breast cancer group was lower than that of control group the difference was statistically significant  $t=4.321 P<0.05$  and the expression of miR 34a in peripheral blood of patients with different T stage N stage clinical stage Ki 67 expression and molecular type was significantly different  $P<0.05$  the expression levels of bcl 2 CCND1 Notch1 in breast cancer lesion of patients with miR 34a median were lower than those of patients with miR 34a <median in breast cancer group  $t=3.660 P<0.05$  the expression level of miR 34a in peripheral blood of patients without pCR after neoadjuvant chemotherapy in breast cancer group was lower than that of patients with PCR the difference was statistically significant  $t=3.660 P<0.05$  ROC curve analysis showed that the expression of miR 34a in

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peripheral blood had predictive value for pCR of neoadjuvant chemotherapy. Conclusion the low expression of miR 34a in peripheral blood of breast cancer patients relates with the pathological characteristics and the efficacy of neoadjuvant chemotherapy detection of miR 34a before chemotherapy has predictive value for pCR.

KEY WORDS Breast cancer Neoadjuvant chemotherapy Pathological complete response miR 34a Prediction

20%  
 1 2  
 sponse pCR  
 pathologic complete re  
 Meta  
 pCR 5  
 pCR 3  
 pCR  
 pCR

RNA microRNA miR  
 4 6 miR 34a  
 RNA miR 34a  
 7 miR 34a  
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 miR 34a  
 pCR pCR

1  
 1.1  
 2017 5 2019 5

B~ TE TEC

142 34-64  
 51.32±9.29 BMI 23.12±6.23 kg/m<sup>2</sup>  
 86 33 ..32

2

2.1 miR 34a  
miR 34a  
P<0.05

2.2  
miR 34a

miR 34a  
P>0.05 T N Ki 67  
miR 34a  
P<0.05 1  
1 miR 34a

Table 1 Comparison of miR 34a expression in peripheral blood of patients with different pathological characteristics in breast cancer group

	n	miR 34a	t/F	P
T	<50	61	0.75±0.22	1.489 0.139
	50	81	0.70±0.18	
	84	0.74±0.21	1.449 0.150	
	58	0.69±0.19		
N	T1 2	101	0.77±0.25	3.991 0.000
	T3 4	41	0.60±0.17	
Ki 67	N0 1	109	0.76±0.24	3.591 0.001
	N2 3	33	0.60±0.16	
	IIB	95	0.77±0.25	
III	47	0.62±0.15		
Luminal HER2		31	0.47±0.16	6.993 <0.001
		111	0.79±0.24	
		63	0.75±0.24	
	38	0.68±0.25		
	41	0.46±0.14		

2.3 miR 34a  
bcd 2 CCND1 Notch1  
miR 34a  
miR 34a  
bcd 2 CCND1 Notch1  
miR 34a  
CCND1 Notch1  
miR 34a  
P<0.05

2.4 pCR pCR miR 34a  
pCR pCR  
miR 34a 0.66±0.16

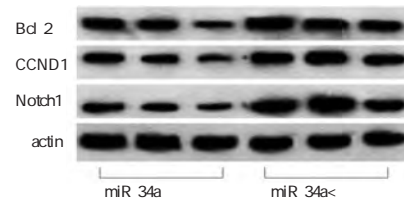


Figure 1 Protein bands of bcl 2 CCND1 Notch1 in breast cancer lesion of patients with different miR 34a expression level in breast cancer group

Table 2 Comparison of bcl 2 CCND1 Notch1 expression levels in breast cancer lesion of patients with different miR 34a expression level in breast cancer group

miR 34a	n	Bcl 2	CCND1	Notch1
	71	0.67±0.16	0.58±0.12	0.55±0.14
<	71	1.14±0.32	0.94±0.27	1.22±0.36
t		9.938	7.582	13.274
P		0.000	0.000	0.000

pCR miR 34a 0.85±0.17  
t=3.660 P<0.05

2.5 miR 34a pCR ROC  
miR 34a pCR ROC  
0.807 95%CI 0.729-0.886 P<0.05

miR 34a pCR  
0.735 82.93%

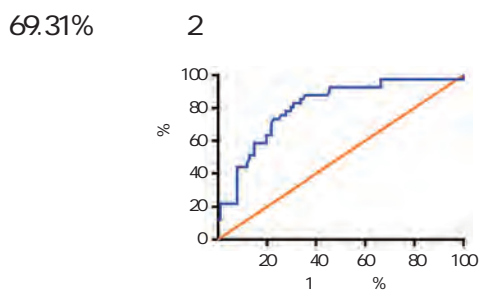
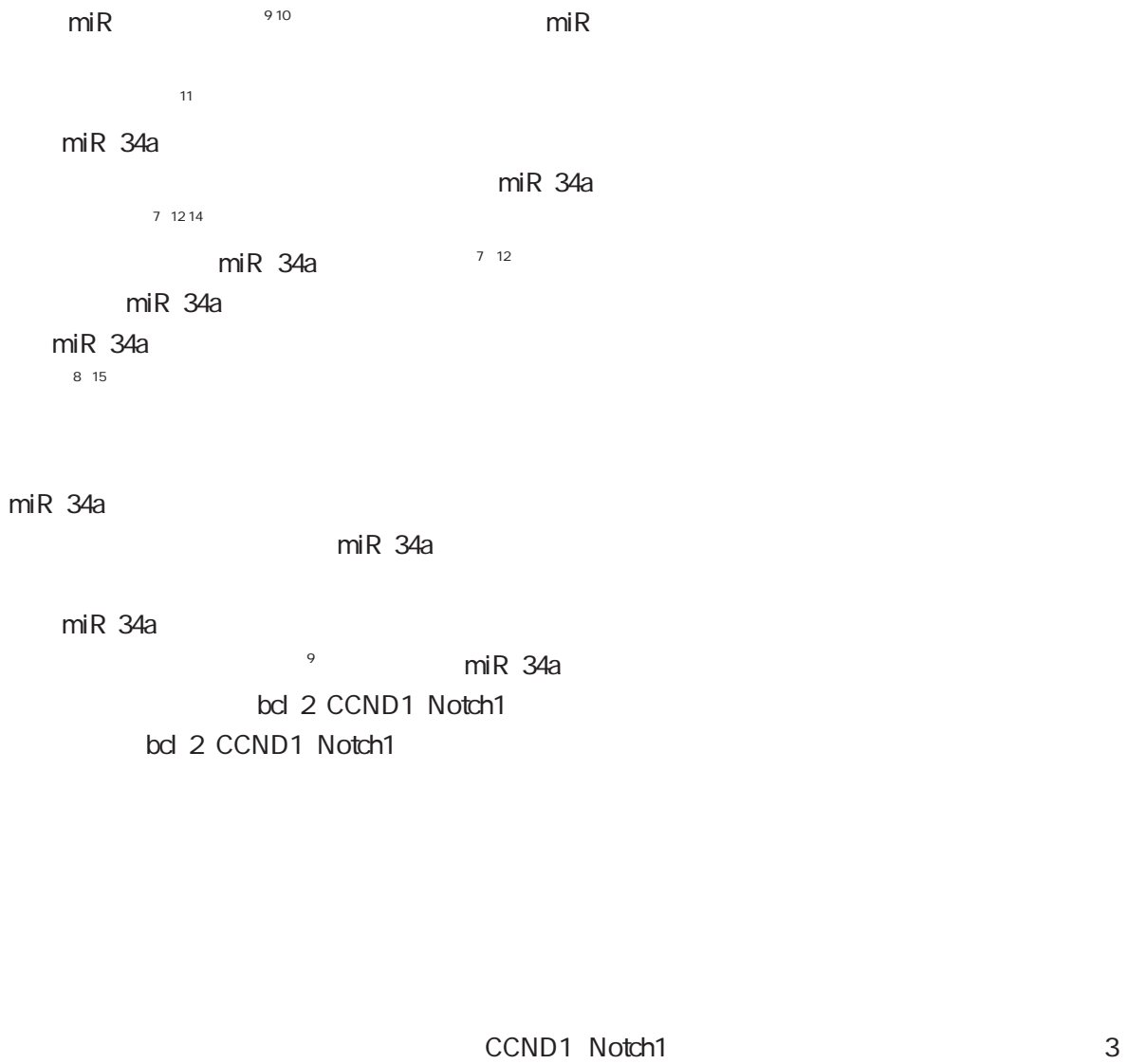


Figure 2 ROC curve of miR 34a in predicting pCR of neoadjuvant chemotherapy

3  
pCR  
miR





expression between groups was analyzed. Multivariate logistic regression analysis was used to explore miR 21 and other detection indicators as the risk prediction of glucose metabolism disorders and diabetes in abdominal obesity patients and ROC curve Assessment. Results The age and ALT of each subgroup were not statistically significant  $P>0.05$ . The waist circumference WHtR and BMI of the T2DM AO group and NC AO group were significantly higher than those of the T2DM NAO group and the NC NAO group and the difference was statistically significant  $P<0.05$ . The T2DM AO group has the highest TG FBG GHbA1C and miR 21 indicators followed by the T2DM NAO group which is evenly and significantly different from the NC AO group and the NC NAO group  $P<0.05$ . There was no difference in the relative expression of miR 21. The stepwise multivariate logistic regression analysis showed that the increased relative expression of miR 21 was the most significant risk factor for diabetes in patients with simple abdominal obesity and the difference was statistically significant  $P<0.05$ . Conclusion The measured expression of miR 21 in peripheral blood of patients with abdominal obesity has important predictive value for the risk of diabetes. Abdominal obese people with higher levels of peripheral blood miR 21 are more at risk of developing diabetes.

KEY WORDS Abdominal obesity Diabetes mellitus miR 21 Logistic regression analysis

ADA

2016 / 0.50 8  
 2 / 24 9

RNA microRNA miR  
 14  
 5 miR 21 1.2  
 67 miR 21 1.2.1  
 12 h  
 Body Mass Index BMI  
 Waist to Height Ratio WHtR  
 Triglyceride TG  
 Fasting Blood Glucose FBG  
 Glycosylated Hemoglobin GHbA1C  
 1.2.2 miR 21  
 1.1  
 2017 1 2019 12  
 160 T2DM 41-79 2  
 52.29±14.03 104 56  
 80 NC 40-  
 79 52.33±14.15 49 31  
 >0.5 T2DM  
 NC T2DM  
 T2DM AO =80 T2DM  
 T2DM NAO =80 NC NC AO  
 =40 NC NC NAO =40  
 3 mL  
 RNA miRNA cDNA  
 miRNA First Strand cDNA Synthesis Kit  
 cDNA cDNA PCR  
 miR 21 5  
 GATCCTAGCATCGTAGCTA 3  
 U6 5 TAGATCGTATAGC  
 TAT 3 5 TAAGCTAGCTAGCTAGC

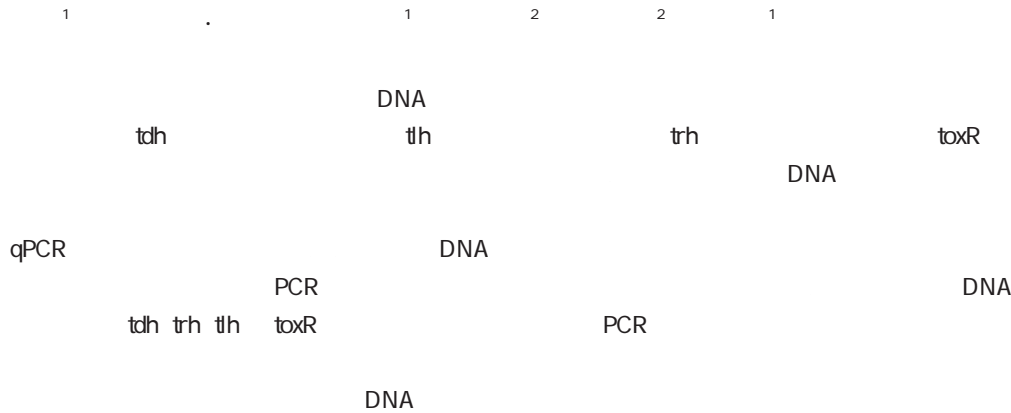
TA 3 95 3min 1 95  
15 s - 60 30 s 40 miRNA  
PCR miRNA Real Time PCR  
Assay kit Ct U6  
2<sup>ct</sup> miR 21  
1.2.3  
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m1 \$ a Y S P<0.05

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10  
10 50% 11 2  
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80 5 12 2  
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18 miR 21  
miR 21  
19 Treg 20 miR 21 Th17/  
IL 10 IL 17 IL 23 IL 6 TGF  
miR 21  
miR 21

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# DNA



## Preparation of plasmid DNA reference material for *Vibrio parahaemolyticus*

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**ABSTRACT** Objective To develop a plasmid DNA reference material including the target genes commonly used for detection of *Vibrio parahaemolyticus* *tdh* *trh* *tlh* and *toxR* gene sequences to provide relevant technical support for the rapid identification of pathogens of *Vibrio parahaemolyticus*. Method The artificial DNA synthesis technology was used to synthesize the desired gene construct a recombinant plasmid and sequence the genes using ultraviolet spectrophotometry a number of laboratories jointly set the value of its purity uniformity stability standard and the uncertainty were checked and the application of real time fluorescence quantitative polymerase chain reaction qPCR to its nucleic acid detection was evaluated. Result The plasmid DNA reference material has high purity good uniformity and stability and can be used to detect a variety of real time fluorescent quantitative polymerase chain reactions of *Vibrio parahaemolyticus* with reliable results. Conclusion The plasmid DNA reference material is suitable for real time fluorescence quantitative PCR detection with *tdh* *trh* *tlh* and *toxR* genes as targets provides technical support for the qualitative and quantitative detection of pathogens and ensures the reliability and comparability of the detection results.

**KEY WORDS** *Vibrio parahaemolyticus* Plasmid DNA standard material Quantitative Real time PCR

*Vibrio parahaemolyticus* VP

VP

1

VP

2017A040405043

S2019154

1.

510515

2.

510623

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E mail xuly@iqtc.cn

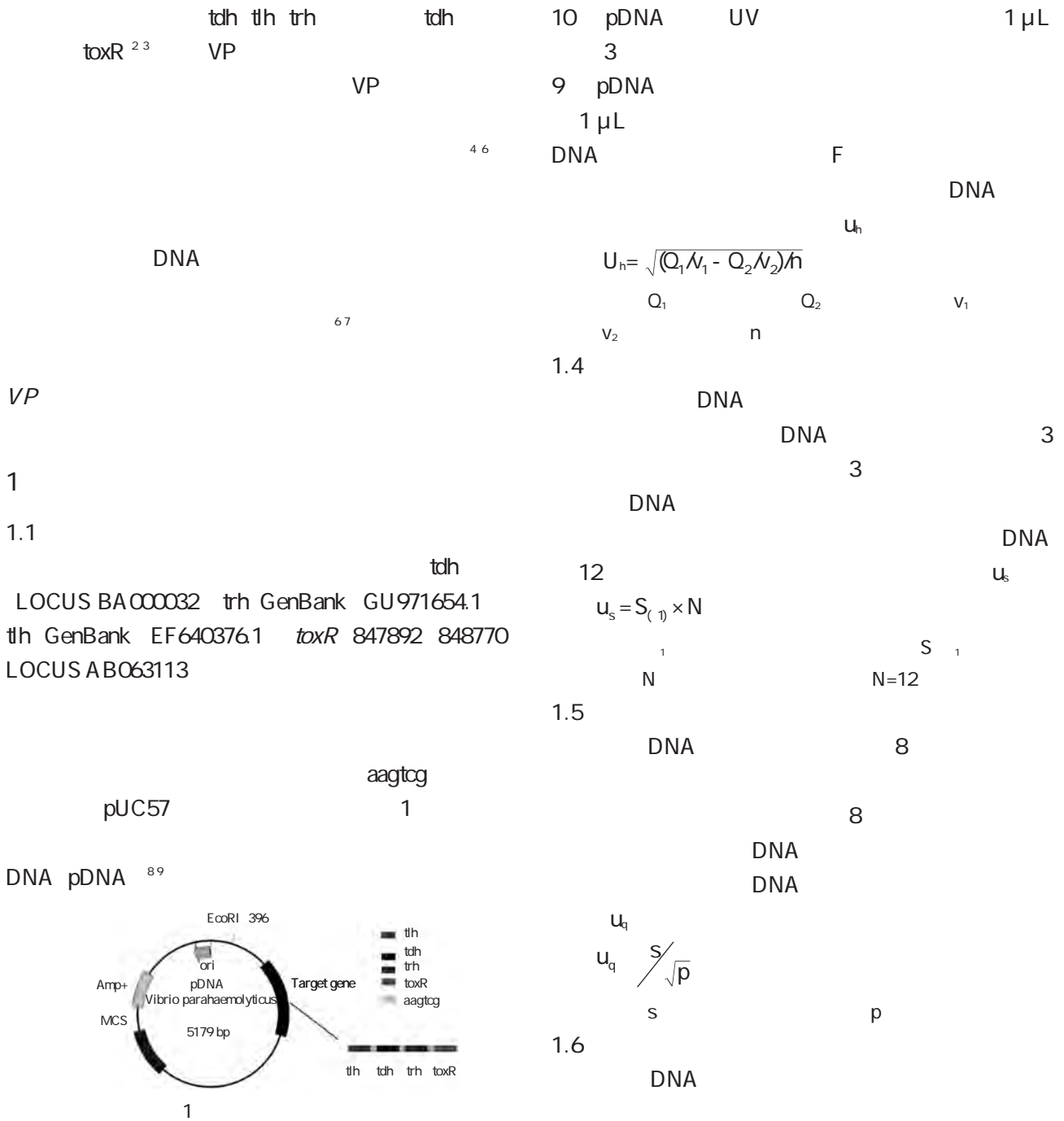


Figure 1 Map of plasmid reference material

1.2

1.3

6

1.7 qPCR

VP DNA gDNA  
 DNA gDNA  
 $\text{copies}/\mu\text{L} = 6.02 \times 10^{23} \text{ copies/mol} \times \text{DNA mass concentration g}/\mu\text{L}$   
 $660 \times \text{DNA size bp g/mol}$   
 3.29 Mbp  
 gDNA 5 179 bp  
 DNA 10  $2 \times 10^6$   
 $2 \times 10^5$   $2 \times 10^4$   $2 \times 10^3$   $2 \times 10^2$   $2 \times 10^1$  copies/mL  
 DNA gDNA qPCR  
 PCR 94 10 min  
 94 30s 58 45s 40 1

1 tdh trh tlh toxR

Table 1 Primer and amplicon for tdh trh tlh and toxR

tdh	5 GGCTGACATCCTACATGACTG 3	83 bp
	5 AGAATGACCGTGCTTATAGCC 3	
trh	5 CGGTCAATCGGTTTTCAACAAC 3	97 bp
	5 AGAAAGAGCTGCCATCGTATAG 3	
tlh	5 TGTTTCGAGACGCTAACTTCTG 3	149 bp
	5 AAATTCTCAGCACCAGACG 3	
toxR	5 AGCAGTACGCAAATCGGTAG 3	121 bp
	5 CAATCGTTGAACCAGAAGCG 3	

1.8

DNA qPCR  
 Limit of detection LOD Limit of  
 quantification LOQ qPCR  
 e K  
 e K gDNA

1.9

SPSS 12.0 Graphpad 5.0

pDNA  
 t pDNA gDNA  
 P<0.05

2

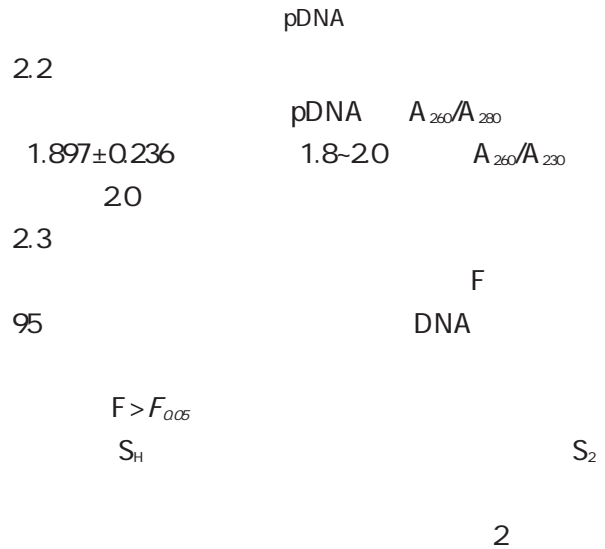
2.1

tdh trh tlh

toxR DNA  
 pUC57 pDNA pDNA  
 100 2

2 DNA

Figure 2 Sequencing diagram of recombinant plasmid



2 pDNA

Table 2 Statistic results of homogenous test of pDNA VP

Q <sub>1</sub>	Q <sub>2</sub>	F	F <sub>0.05</sub>
2.64	72.29	0.49	F <sub>0.05</sub> (2,27)3.35
29.06	16.93	3.80	F <sub>0.05</sub> (9,20)2.39

1 u<sub>i</sub>  
 0.892 μg/mL

2.4

12 pDNA VP  
 pDNA

VP  $Y = 1 + 0$  A  
 $n = 2$   $P = 0.95$  95% t  
 0.183  $|t_{0.95, n-2}| S_{1, n-2}$   
 $S_{bl}$  3

3 pDNA B  
 Table 3 Statistic results of short term stability of pDNA VP

	1	0	S	$t_{0.95, n-2}$	S
	0.032	29.959	0.101	0.183	

DNA 12 C  
 $u_k$   $u_k$   
 1.212  $\mu\text{g/mL}$

2.5 D  
 8  
 DNA  
 $s^2 = 29.62 \mu\text{g/mL}$   
 DNA

$u_k$  3 pDNA tdl trh tlh toxR  
 $u_k$  0.296  $\mu\text{g/mL}$   
 2.6

Figur 3 Standard curve of tdl trh tlh and toxR established by pDNA VP

DNA  
 $u_s$   $u_h$   $u_k$   
 DNA  
 $U_{CRM}$   $U_{cm}$   
 1.534  $\mu\text{g/mL}$   $3.068 \mu\text{g/mL}$

4 pDNA  
 Table 4 Data for the standard curve established by pDNA VP

		$R^2$	%	LOD copy/mL	LOQ copy/mL
torx	$y = 40.75 - 3.337x$	0.997	94.984 450	$10^1$	$10^1$
tlh	$y = 39.574 - 3.649x$	0.999	86.639 133	$10^1$	$10^1$
tdl	$y = 36.675 - 3.58x$	0.999	90.252 165	$10^1$	$10^1$
trh	$y = 40.708 - 3.643x$	0.998	88.147 754	$10^1$	$10^1$

2.7 qPCR  
 $k = 2$  3  
 DNA  
 qPCR  
 4

2.8 DNA  
 DNA gDNA  
 95  
 gDNA DNA  
 $P < 0.05$  5

10 11 H7

3 DNA  
 12 13

trh th toxR  
- 20

DNA  
tdh

DNA DNA

DNA

13 14

DNA

tdh trh th toxR

DNA

DNA

1

2

8 941 943

3 . 82

J . 2018 38

1 37 40

4

J . 2007 27

7 664 668

5

PCR

J .

2011 36 4 22 27.

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8 DNA

J . 2014 40 z1 9 13

9

J . 2019 23 3 192 199.

10

J . 2017 29

4 273 276 280

11

J . 2016 47 4 605 608.

12

. H7

RT PCR J .

2014 50 11 13 15 18

13

. 273 < ž

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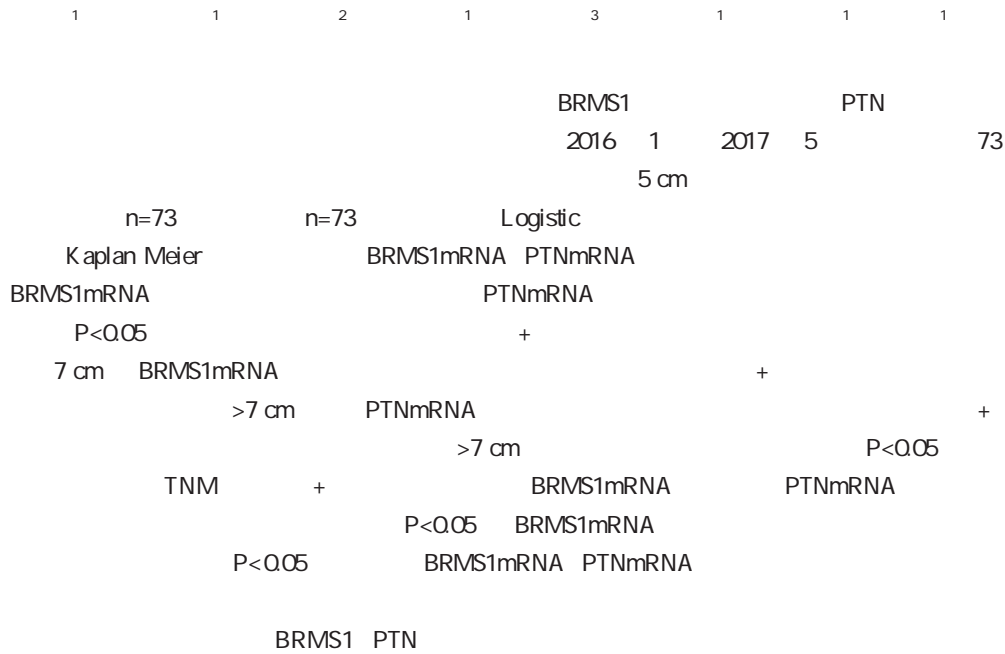
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J .

.132

q4440Y

## BRMS1mRNA PTNmRNA



### Expression and clinical significance of *BRMS1*mRNA and *PTN*mRNA in patients with rectal cancer

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adjacent normal tissue group n=73 . Multivariate Logistic regression was used to analyze the risk factors that affect the prognosis of patients with rectal cancer. Kaplan Meier survival curve was drawn to study the effect of BRMS1mRNA and PTNmRNA on the prognosis of patients. Results The expression level of BRMS1mRNA in the rectal cancer tissue group was lower than that in the normal tissues adjacent to the cancer and the positive rate of PTNmRNA was higher than that of the normal tissues adjacent to the cancer the differeg

gl

Eppendoff  
M MLVcDNA  
RNase free  
BRMSI 3  
RT PCR  
Trizol RNA  
PCR  
GAPDH  
BRMSI GAPDHcDNA

BRMS1mRNA PTNmRNA  
Logistic  
P<0.05  
2  
2.1 BRMS1mRNA PTNmRNA

Elivision TM plus  
PTN PTN PTN  
SantaCruz

BRMS1mRNA  
PTNmRNA  
P<0.05 1  
1 BRMS1mRNA PTNmRNA

Leica  
1.3.2 BRMS1mRNA PTNmRNA  
BRMS1mRNA BRMSI  
5 GACCGCCAGAGCCrIG GA 3 5  
CTGCCTCTGGCGTGCAG 3 5 FAM  
CAGCTCTGAArGGTGG MGB 3 GAPDH  
5 CATCAATGACCCCTIIG 3  
5 CATGGGTGGAATCATATTFGGAAC 3

Table 1 Comparison of BRMS1mRNA and PTNmRNA expression in 2 groups n % -

	n	BRMS1mRNA	PTNmRNA
	73	0.59±0.06	59 80.82
	73	0.87±0.07	21 28.77
<sup>z</sup> t		25.948	39.929
P		<0.001	<0.001

5 CATGGGTGGAATCATATTFGGAAC 3  
5 VIC CCTCAACTACATGGTITAC MGB  
3 50-100µg Trizol RNA  
RT PCR  
c DNA 37 1 h 95 10 min  
MMLV PCR 94 1 min  
58 1 min 72 1 min 35 72  
7 min 1.5%  
BRMS1 action mRNA  
PTNmRNA

2.2 BRMS1mRNA PTNmRNA  
BRMS1mRNA PTNmRNA  
+  
7 cm BRMS1mRNA  
+  
>7 cm  
PTNmRNA  
+  
>7 cm

PBS  
1.4

P<0.05 2  
2.3

3 2020 5 31  
2 36  
29.98±2.64  
overall Survival OS  
6

73 49  
24 32.88%  
TNM +  
BRMS1mRNA  
PTNmRNA  
P<0.05 3

1.5  
SPSS 18.0  
n %  
t Kaplan Meier

2.4 BRMS1mRNA PTNmRNA  
BRMS1mRNA PTNmRNA  
Keplan meier 1

2 BRMS1mRNA PTNmRNA

Table 2 Relationship between BRMS1mRNA PTNmRNA and different pathological parameters in patients with rectal cancer

		n=73	BRMS1mRNA	t	P	PTNmRNA	n=59	<sup>2</sup>	P
TNM	<55	41	0.73±0.12	0.756	0.435	34	57.63	0.002	0.961
		32	0.71±0.09			35	59.32		
		35	0.69±0.15	1.830	0.071	27	45.76	0.587	0.444
		55	0.63±0.13			32	54.24		
		48	0.71±0.19			35	59.32		
	+	25	0.59±0.13	3.191	0.002	24	40.68	7.239	0.007
		50	0.76±0.14			37	62.71		
		23	0.53±0.16	6.232	<0.001	22	37.29	4.765	0.029
		45	0.87±0.16			31	52.54		
		28	0.51±0.13			28	47.46		
	+	40	0.89±0.32	7.513	<0.001	28	47.46	6.686	0.010
		33	0.43±0.16			31	52.54		
		52	0.86±0.13	10.977	<0.001	39	66.10	3.953	0.047
		21	0.53±0.07			20	33.90		
		cm	7	39	0.73±0.28	4.093	<0.001	27	45.76
>7	34		0.49±0.21	32	54.24				

3

Table 3 Single factor and multiple factor analysis of prognostic survival in patients with rectal cancer

		OR	95%CI	P	OR	95%CI	P
TNM	>7 cm vs 7 cm	0.764	0.664	0.594	-	-	-
	vs	0.849	0.168	0.016	0.835	0.146	<0.001
	vs	0.823	0.669	0.638	-	-	-
	+ vs +	0.794	0.131	0.009	0.893	0.189	<0.001
	vs	0.726	0.189	0.013	0.724	0.163	<0.001
BRMS1mRNA	vs	0.971	0.261	0.045	0.789	0.261	<0.001
PTNmRNA	vs	0.879	0.159	0.033	0.894	0.127	<0.001

BRMS1L  
BMRS1  
Cx43  
BRMS1  
13  
BRMS1L  
BRMS1mRNA  
PTN  
14 PTN  
MDK  
PTN  
PTN  
15  
PTN  
Wang 16

‘ . .  
UfX, !r•QÐ" Ö• †Ð @& " N †YÐ»p°Ã % UD P

Treg

Treg

cervical cancer

1.2

3.5 T regulatory T cell Treg

CD4+T

Foxp3

10 interleukin 10 IL 10

transforming growth factor 1 TGF 1

6.8

Treg Foxp3

IL 10 TGF 1

1

1.1

2015 3 2018 12

CIN

CIN

84

38-62

47.62±7.23 CIN 38 33-59

46.48±8.65 60 38-

64 48.14±9.14 CIN

P>0.05

1.2

RIPA

BCA

SantaCruz

Elisa

Bio rad

1.3

1.3.1 Foxp3 cydinD1 p53

CIN

RIPA

BCA

30 µg

5% NC 4 1 1 000

Foxp3 cydinD1 p53 actin

NC ECL actin

1.3.2 IL 10 TGF 1

CIN

RIPA

Elisa

IL 10 TGF 1

1.4

SPSS 20.0

- t

P<0.05

2

2.1 Foxp3 IL 10 TGF 1

CIN

Foxp3 IL 10 TGF 1

P<0.05 CIN

Foxp3 IL 10 TGF

1 P<

0.05 1 1

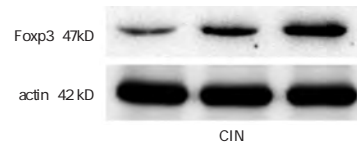


Figure 1 Protein bands of Foxp3 in cervical cancer CIN and normal cervical tissues

Table 1 Comparison of Foxp3 expression IL 10 and TGF 1 Contents in each groups

	n	Foxp3	IL 10 ng/mL	TGF 1 ng/mL
	60	0.93±0.15	0.83±0.17	0.74±0.20
CIN	38	1.44±0.20 <sup>a</sup>	1.51±0.32 <sup>a</sup>	1.32±0.28 <sup>a</sup>
	84	1.89±0.41 <sup>ab</sup>	2.11±0.52 <sup>ab</sup>	1.89±0.44 <sup>ab</sup>
F		189.938	237.751	203.475
P		0.000	0.000	0.000

<sup>a</sup>P<0.05 CIN <sup>b</sup>P<0.05

2.2 Foxp3 IL 10 TGF 1 IL 10 TGF 1  
 Foxp3 IL 10 TGF 1 P>0.05 HPV FIGO ~  
 r 0.351 0.283 P<0.05 Foxp3  
 2.3 Foxp3 IL 10 TGF 1 HPV  
 IL 10 TGF 1 FIGO ~  
 Foxp3 P<0.05 2  
 2 Foxp3 IL 10 TGF 1 -

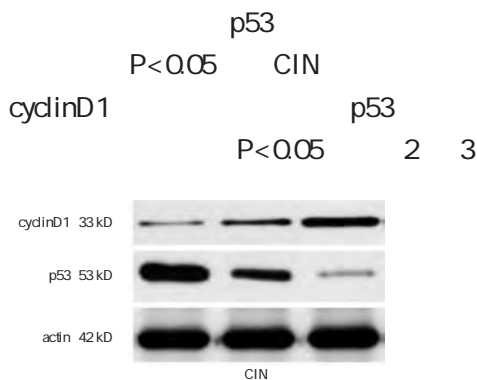
Table 2 Comparison of Foxp3 expression, IL 10 and TGF 1 Contents in cervical cancer tissues with different clinicopathological characteristics -

	n	Foxp3	IL 10 ng/mL	TGF 1 ng/mL
<40	34	1.95±0.48	2.06±0.48	1.86±0.51
40	46	1.88±0.37	2.17±0.60	1.93±0.36
t		0.737	0.886	0.720
P		0.463	0.378	0.474
<3 cm	28	1.99±0.51	2.02±0.42	1.85±0.34
3 cm	52	1.85±0.35	2.19±0.66	1.95±0.52
t		1.448	1.233	0.916
P		0.152	0.221	0.362
HPV	18	1.51±0.34	1.67±0.38	1.45±0.34
	62	2.21±0.62	2.58±0.61	2.32±0.51
t		4.580	5.985	6.796
P		0.000	0.000	0.000
FIGO	~	46	1.58±0.31	1.75±0.33
	~	34	2.19±0.59	2.51±0.57
t		5.990	7.509	6.520
P		0.000	0.000	0.000
	33	2.13±0.66	2.60±0.59	2.26±0.50
	47	1.64±0.28	1.62±0.42	1.51±0.37
t		4.549	8.685	7.713
P		0.000	0.000	0.000
	48	1.67±0.33	1.61±0.29	1.40±0.30
	32	2.14±0.62	2.63±0.63	2.37±0.56
t		4.407	9.790	10.050
P		0.000	0.000	0.000

2.4 cydinD1 p53 3 cydinD1 p53 -  
 Table 3 Comparison of CyclinD1 and p53 expression in each groups -

	n	cydinD1	p53
CIN	60	0.92±0.16	1.05±0.23
	38	1.26±0.20 <sup>a</sup>	0.55±0.08 <sup>b</sup>
	84	2.02±0.42 <sup>ab</sup>	0.22±0.05 <sup>ab</sup>
F		175.582	332.842
P		0.000	0.000

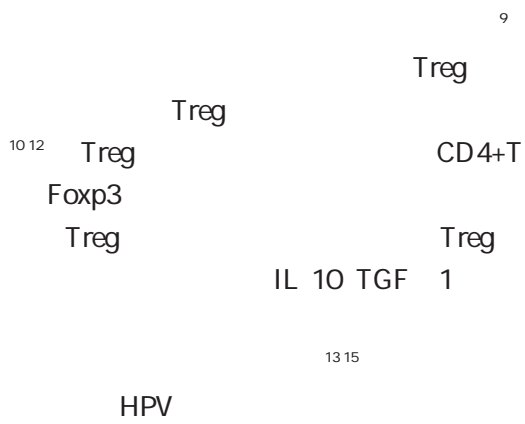
<sup>a</sup>P<0.05 CIN <sup>b</sup>P<0.05



2 CIN cydinD1 p53  
 Figure 2 Protein bands of CyclinD1 and p53 in cervical cancer, CIN and normal cervical tissues

2.5 Foxp3 IL 10 TGF 1  
 cydinD1 p53  
 Foxp3 IL 10 TGF 1  
 CydinD1 p53  
 4

3



# PG MG7 Ag G 17

PG MG7 Ag G 17

2018 6 2019 1

126 n=36 51 n=38 n=38 n=46 n=38 n=52

n=28 PG MG7 Ag G 17 HP

P<0.05 PG PGR MG7 Ag G 17 PGR

G 17 P<0.05 MG7 Ag P<0.05

PG PG PGR P>0.05 G 17

PGR PG PG P

P<0.05 HP P<0.05 PG+G 17+MG7

Ag+HP P<0.05

PG MG7 Ag G 17

PG MG7 Ag G 17

## Diagnosis and differential diagnosis of PG MG7 Ag combined with G 17 detection for gastric precancerous lesions and gastric cancer

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 Health Management Center the First People s Hospital of Chenzhou City Hunan Province Chenzhou Hunan China 423000

**ABSTRACT** Objective To analyze the diagnostic value of pepsinogen PG MG Antigen7 Ag MG7 Ag combined with gastrin 17 Gastrin 17 G 17 detection for gastric precancerous lesions and gastric cancer. Methods 126 patients with gastric cancer who met the inclusion criteria were collected from June 2018 to January 2019 and set as the gastric cancer group. According to the different differentiation levels of histopathological tumor cells they will be divided into a highly differentiated group n=38 and a moderately differentiated group n=52 and poorly differentiated group n=36 . At the same time 51 cases with normal healthy physical examination in this hospital healthy control group and 112 cases with gastric precancerous lesions superficial gastritis group were selected. Patients in the gastric precancerous lesion group were again divided into superficial gastritis group n=38 gastric ulcer group n=46 and chronic atrophic gastritis group

n=28 . The indicator levels of PG MG7 Ag and G 17 in different populations were compared the HP positive infection rate was calculated and the diagnostic efficiency of different tests for gastric precancerous lesions and gastric cancer were compared. Results The expression levels of MG7 Ag and G 17 in the gastric cancer group were higher than those in the gastric precancerous lesion group and the healthy control group P<0.05 . The expression levels of PG and PGR in the gastric precancerous lesion group were significantly higher than those in the healthy control group and gastric cancer group. The PGR level in the gastric cancer group was significantly lower than that in the gastric precancerous lesion group and the healthy control group. The difference between the groups was statistically significant P<0.01 . MG7 Ag in the low differentiation group was higher than that in the high differentiation group and moderate differentiation group. and the G 17 level in the high differentiation group was significantly lower than that in the moderate differentiation group and low differentiation group P<0.05 . There was no significant difference in the levels of PG PG and PGR among patients with high medium and low differentiation of gastric cancer P>0.05 . The levels of G 17 PGR and PG in the chronic atrophic gastritis group were significantly lower than those in the superficial gastritis group and gastric ulcer group. The PG in the superficial gastritis group was low than that in the other two groups P<0.05 . The positive rate of HP in the gastric cancer group was higher than in the gastric precancerous lesion group P<0.05 . The specificity and sensitivity of PG +G 17+MG7 Ag+HP in detecting gastric cancer superficial gastritis gastric ulcer and chronic atrophic gastritis were significantly higher than those of single test P<0.05 . Conclusion PG MG7 Ag and G 17 are related to the occurrence and progression of gastric cancer. The detection of the above indicators can effectively evaluate the patient s condition and the combined detection can improve the sensitivity and specificity of diagnosis.

KEY WORDS PG MG7 Ag G 17 Gastric precancerous lesions Gastric cancer

				126			
		25.2/10				n=38	
32.8/10	17/10		n=52		n=36	78	
23.2% <sup>1</sup>			48	27~71		48.28±6.11	
				>18			
	2					7	
			helico			112	
bacter pylori HP		HP			64	48	
				22~70		46.96±8.07	
	pepsinogen PG						
MG antigen7 Ag MG7 Ag		17 gastrin 17	38	46		28	
G 17					>18	1	
			34				
	PG+MG7 Ag+G 17				55		
1					33	22	22~70
1.1					47.25±7.53	3	
	2018 6	2019 1					
					P>0.05		

1.2 SPSS 18.0  
 1.2.1 n %<sup>2</sup> -  
 3 PG MG7 Ag t P<  
 G 17 HP 0.05  
 2  
 1.2.2 2.1 PG MG7 Ag G 17  
 3-5 mL 2.1.1 3 PG MG7 Ag G 17  
 - 20  
 Biohit ELISA PG PG MG7 Ag G 17  
 G 17 MG7 Ag PG /PG  
 PGR P<0.05 PG PGR  
 MG7 Ag>3 U/mL PGR  
 13/14 HP  
 P<0.05 1  
 1 3 PG MG7 Ag G 17 -

Table 1 Comparison of the expression levels of PG MG7 Ag and G 17 among 3 groups -

	n	PG μg/mL	PGII μg/mL	PGR	MG7 Ag U/mL	G 17 pmol/L
	51	116.36±30.25	8.49±3.26	12.63±5.41	1.43±0.50	8.36±4.02
	112	140.32±52.02 <sup>a</sup>	14.25±4.82 <sup>a</sup>	14.36±6.25 <sup>a</sup>	1.52±0.45	13.25±5.02 <sup>a</sup>
	126	77.66±34.51 <sup>ab</sup>	20.78±9.33 <sup>ab</sup>	3.94±1.59 <sup>b</sup>	2.91±1.07 <sup>ab</sup>	23.15±9.74 <sup>ab</sup>
F	-	68.29	62.31	164.62	114.99	93.61
P	-	<0.001	<0.001	<0.001	<0.001	<0.001

<sup>a</sup> P<0.05 <sup>b</sup> P<0.05

2.1.2 PG MG7 Ag G 17 P<0.05  
 PG PG PGR  
 MG7 Ag P>0.05 2  
 G 17

2 PG MG7 Ag G 17 -

Table 2 Comparison of expression levels of PG Mg7 AG and G 17 in gastric cancer patients with different degrees of differentiation -

	n	PG μg/mL	PG μg/mL	PGR	MG7 Ag U/mL	G 17 pmol/L
	38	77.45±29.63	23.84±7.46	3.46±2.01	2.51±0.56	21.25±4.15
	52	81.15±31.69	21.69±9.11	3.51±2.13	2.93±0.71 <sup>a</sup>	25.36±7.42 <sup>a</sup>
	36	75.78±30.45	17.36±9.59	3.94±2.39	3.26±0.68 <sup>a</sup>	27.63±10.51 <sup>a</sup>
F	-	0.36	5.22	0.56	12.06	6.59
P	-	0.700	0.006	0.572	0.004	0.001

<sup>a</sup> P<0.05

2.1.3 PG MG7 Ag G 17 P<0.05 3  
 2.2 HP  
 G 17 PGR PG HP HP  
 PG P<0.05 HP  
 4

3 PG MG7 Ag G 17  
 Table 3 Comparison of expression levels of PG Mg7 AG and G 17 in patients with different precancerous diseases

	n	PG µg/mL	PG µg/mL	PGR	MG7 Ag U/mL	G 17 pmol/L
	38	119.65±41.08	10.05±4.39	12.71±5.36	1.56±0.31	10.82±4.51
	46	170.85±43.15 <sup>a</sup>	20.47±6.49 <sup>a</sup>	7.15±3.54 <sup>a</sup>	1.86±0.53 <sup>a</sup>	14.96±6.10 <sup>a</sup>
	28	80.49±31.69 <sup>a</sup>	17.48±7.15 <sup>a</sup>	5.09±1.32 <sup>a</sup>	1.62±0.21 <sup>b</sup>	4.39±2.07 <sup>ab</sup>
F	-	46.97	31.72	35.26	6.59	41.69
P	-	<0.001	<0.001	<0.001	0.002	<0.001

<sup>a</sup> P<0.05 <sup>b</sup> P<0.05

4 HP n %  
 Table 4 HP infection in different populations n %

	n	HP
	112	64 57.14
	38	16 42.10
	46	36 78.26
	28	12 42.85
<sup>2</sup>	-	14.219
P	-	<0.001
	126	108 85.71
	38	32 84.21
	52	44 84.61
	36	32 88.88
<sup>2</sup>	-	0.418
P	-	0.811

2.3 PG+G 17+MG7 Ag+HP

P<0.05 5

3

90%

Endo <sup>5</sup> HP

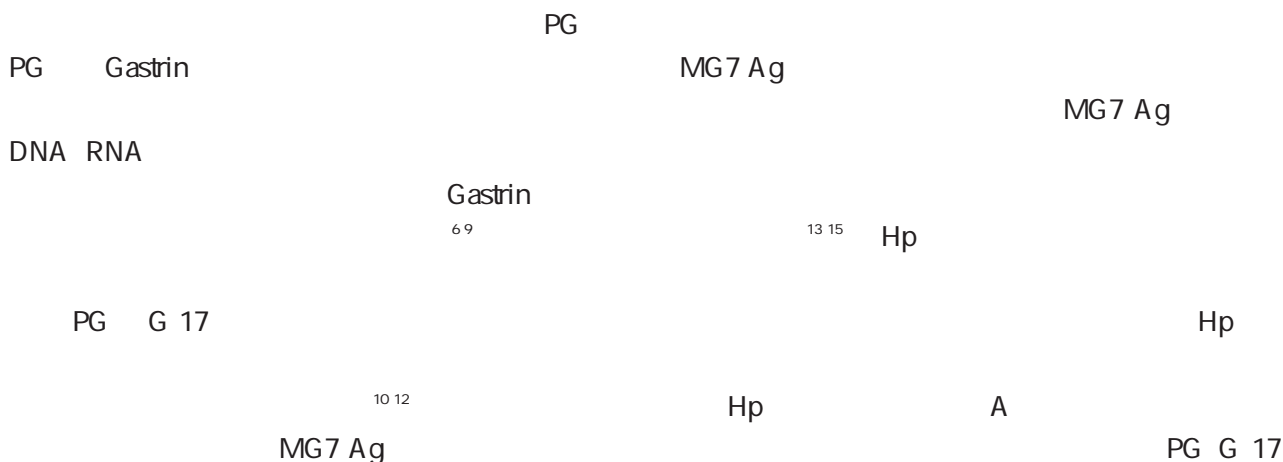
HP

PG

5 %  
 Table 5 Diagnostic efficacy of combined diagnosis for precancerous lesions and gastric cancer %

	n=63	n=19	n=23	n=14
HP	39.68 <sup>a</sup>	42.85 <sup>a</sup>	52.63 <sup>a</sup>	57.14 <sup>a</sup>
PG PG +PG	36.50 <sup>a</sup>	41.26 <sup>a</sup>	47.36 <sup>a</sup>	50.00 <sup>a</sup>
G 17	44.44 <sup>a</sup>	47.61 <sup>a</sup>	42.10 <sup>a</sup>	57.14 <sup>a</sup>
MG7 Ag	57.14 <sup>a</sup>	63.49 <sup>a</sup>	52.63 <sup>a</sup>	64.28 <sup>a</sup>
PG+G 17+MG7 Ag	87.30	85.71	68.42 <sup>a</sup>	73.68 <sup>a</sup>
PG+G 17+MG7 Ag+HP	90.47	88.88	78.94	84.21

<sup>a</sup> PG+G 17+MG7 Ag+HP P<0.05



MG7 Ag

HP

16

PG G 17 HP

PG MG7 Ag G 17

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## A/G NT proBNP PCI

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NT proBNP AMI A/G PCI 2017

1 2019 7 178 AMI AMI

PCI 121 PCI PCI 57 A/G NT proBNP

ABL GLB PCI PIC

logistic A/G

NT proBNP PCI A/G ABL NT proBNP GLB

P<0.05 AMI LVEF CRP C ABL GLB A/G

NT proBNP P<0.05 LVEF

ABL A/G CRP GLB Logistic

65 CRP 45 mg/L AMI PCI LVEF A/G

NT proBNP P<0.05 A/G NT proBNP AMI

PCI NT proBNP PCI

### Analysis of the value of preoperative albumin globulin ratio and NT proBNP in prognosis of patients with acute myocardial infarction after PCI

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**ABSTRACT** Objective To analyze the evaluation value of preoperative albumin globulin ratio A/G and the N Terminal pro brain natriuretic peptide NT proBNP in prognosis of patients with AMI after PCI. Method A total of 178 patients with AMI treated in our hospital from January 2017 to July 2019 were selected as the research objects and all patients met the clinical diagnostic criteria of AMI. Among them 121 patients treated with PCI were selected as the PCI group and 57 patients without PCI treatment were selected as the control group. The expression levels of A/G NT proBNP ABL and GLB were measured and compared between the two groups. Patients with PCI were further divided into groups with good prognosis and poor prognosis according to whether cardiac adverse events occurred after PCI. Logistic multivariate regression analysis was used to analyze the relevant factors affecting its prognosis. Results There were significant

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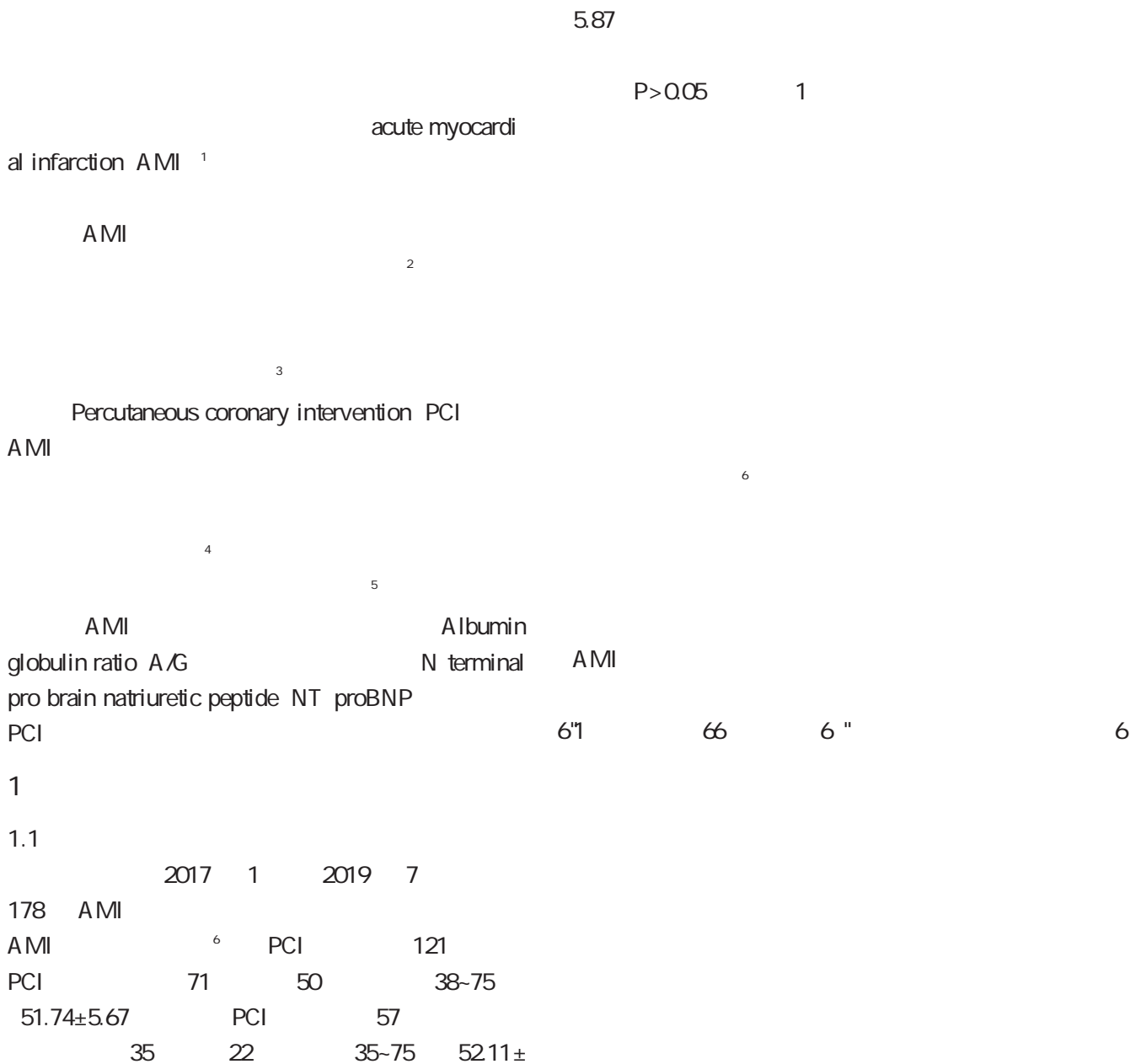
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differences in the expression levels of A/G and NT proBNP between the two groups. The levels of A/G and ABL in the PCI group were higher than those in the control group and the levels of NT proBNP and GLB were lower than those in the control group  $P < 0.05$ . Among patients with different prognosis there were significant differences in age smoking history LVEF CRP ABL GLB A/G and NT proBNP  $P < 0.05$ . The number of patients with good prognosis without smoking history LVEF ABL A/G were higher than those with poor prognosis group and the age levels of CRP and GLB were lower than those with the poor prognosis group. Logistic regression analysis showed that age  $\geq 65$  years and CRP  $\geq 45$  mg/L were independent risk factors for cardiac adverse events after PCI in AMI patients. LVEF A/G and NT proBNP were protective factors for prognosis  $P < 0.05$ . Conclusion The levels of A/G and NT proBNP are related to the prognosis of patients and they are the prognostic factors of AMI patients after PCI which can provide a reference for their postoperative evaluation.

KEY WORDS Acute myocardial infarction Albumin globulin ratio NT proBNP PCI Evaluation value



Left ventricular ejection fraction LVEF <sup>2</sup> PCI  
 1.3 Logistic P<0.05  
 PCI A/G NT proB  
 NP ABL GLB PCI  
 AMI 2  
 2.1 A/G NT proBNP ABL GLB  
 1.4 PCI A/G ABL  
 SPSS 18.0 NT proBNP GLB  
 - n % P<0.05 2  
 2 A/G NT proBNP -

Table 2 Comparison of A/G and NT NT proBNP expression levels between 2 groups -

	A/G(%)	NT proBNP ng/L	ABL g/L	GLB mg/L
PCI n=121	0.25±0.08	1342.11±88.67	38.78±5.63	30.33±4.12
n=57	0.19±0.07	2975.12±174.36	33.68±3.54	34.26±4.72
t	4.853	82.903	6.274	5.662
P	0.000	0.000	0.000	0.000

2.2 AMI CRP 45 mg/L AMI PCI  
 AMI LVEF LVEF A/G  
 CRP ABL GLB A/G NT proBNP NT proBNP  
 P<0.05 P<0.05 4  
 LVEF ABL A/G CRP  
 GLB P<0.05 3 3  
 3 AMI

Table 3 Comparison of clinical data of AMI patients with different prognosis

	n=88	n=33	<sup>2</sup> P	
	65	21	27	33.6820.000
	<65	67	6	0.070 0.792
		51	20	17.4000.000
		37	13	
		27	24	
		61	9	
LVEF %	50	57	12	7.904 0.005
	<50	31	21	
CRP mg/L	45	33	26	16.3750.000
	<45	55	7	
ABL g/L	39	54	13	4.688 0.030
	<39	34	20	
GLB g/L	32	32	29	25.4780.000
	<32	56	4	
A/G	1.19	63	10	11.1250.001
	<1.19	36	23	
NT proBNP ng/L	1340	38	22	7.736 0.005
	<1340	60	11	

2.2 AMI PIC Logistic NT proBNP  
 Logistic 65 11 12

4 AMI PCI Logistic  
 Table 4 Multivariate logistic regression analysis of PCI prognosis in patients with AMI

			Wald/ <sup>2</sup>	OR	95%CI	P
	0.330	0.011	1.163	1.390	1.361~1.421	0.021
	0.151	0.335	0.187	1.162	0.603~2.224	0.631
LVEF	-0.533	0.183	1.231	0.586	0.409~0.840	0.033
CRP	0.555	0.212	4.562	1.741	1.149~2.639	<0.001
ABL	0.151	0.335	2.314	1.162	0.603~2.242	0.112
GLB	0.167	0.331	1.032	1.181	0.617~2.260	0.342
A/G	-0.25	0.05	6.485	0.778	0.706~0.858	<0.001
NT proBNP	-0.411	0.185	6.899	0.662	0.463~0.949	<0.001

NT proBNP ABL  
 NT proBN ABL  
 A/G  
 NT proBN 13 PCI 20  
 PCI A/G NT proBNP  
 NT proBNP AMI PCI  
 65 PCI  
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 J . 2020 46 4  
 19 ABL 427 429. 1500

# caspase 3 P53

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2018 9 2019 6

50 caspase 3 P53

ROC caspase 3 P53

P<0.05

P53 P<0.05 caspase 3 P53 >

caspase 3 P53 caspase 3 P53

14.76 pg/mL 0.835 ng/mL

caspase 3 P53

## Correlation between serum apoptosis molecules caspase 3 p53 and the disease condition and therapeutic effect of sudden deafness

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**ABSTRACT** Objective To study the correlation between serum apoptotic molecules Caspase 3 p53 with the disease condition and the therapeutic effect of sudden deafness. Methods 50 patients with sudden deafness admitted to our hospital from September 2018 to June 2019 were selected as the observation group and 50 healthy volunteers were selected as the control group. The serum levels of Caspase 3 and p53 were detected. The disease condition and curative effect of the observation group were evaluated and the predictive value of Caspase 3 and p53 on curative effect was analyzed by ROC curve. Results The levels of serum caspase 3 and p53 in the observation group were higher than those in the control group the difference was statistically significant  $P<0.05$  and the differences of serum caspase 3 and p53 levels of patients with different degree of hearing impairment and type of hearing curve the difference was statistically significant  $P<0.05$  the total effective rate of patients with Caspase 3 p53 content  $>$  median in the observation group was lower than that in the patients with caspase 3 and p53 content  $\leq$  median the difference was statistically significant  $P<0.05$ . Serum Caspase 3 and p53 levels have predicted value on curative effect the best cut off points 14.76 pg/mL 0.835 ng/mL respectively. Conclusion The increase in serum apoptotic molecules caspase 3 and P53 is related to the occurrence aggravation of the disease and the therapeutic effect of sudden deafness.

**KEY WORDS** Sudden deafness Apoptosis Caspase 3 p53 Curative effect

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 1.1  
 2018 9 2019 6  
 50  
 28 22 37-59  
 47.47±8.95 50  
 30 20  
 35-60 48.32±9.37  
 P>0.05  
 2015 5  
 1.2 caspase 3 P53  
 5-8mL  
 5-8mL  
 3 000 / 10 min  
 caspase 3  
 P53  
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 26-40 d  
 Decibel Hearing Level BHL

41-60 d BHL  
 61-80 d BHL  
 81 d BHL  
 1 k Hz  
 2 k Hz  
 <80 d BHL  
 0.5-4.0 k Hz  
 2015 5  
 30 d BHL  
 BHL  
 1.5  
 SPSS 21.0  
 Prism6.0  
 P<0.05  
 2  
 2.1 caspase 3 P53  
 P53  
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 1 caspase 3 P53  
 Table 1 Comparison of serum caspase 3 and p53 contents between 2 groups  

	n	Caspase 3 pg/mL	P53 pg/mL
	50	17.67±5.29	0.98±0.29
	50	6.58±1.31	0.39±0.10
t		14.389	13.600
P		0.000	0.000

  
 2.2 caspase 3 P53  
 caspase 3  
 P53  
 P<0.05 2  
 2.3 caspase 3 P53  
 caspase 3 P53  
 caspase 3 P53 >  
 P<0.05 3

2 caspase 3 P53

Table 2 Comparison of serum caspase 3 and p53 contents in patients with different diseases condition in the observation group

	n	Caspase 3 (pg/mL)	P53 (pg/mL)
	11	8.95±2.44	0.54±0.14
	19	15.52±4.08	0.79±0.22
	13	22.13±5.77	1.21±0.29
	7	28.93±7.74	1.76±0.35
F		28.391	23.575
P		0.000	0.000
	15	7.58±2.23	0.49±0.14
	18	14.58±3.24	0.80±0.24
	7	26.39±8.85	1.22±0.31
	10	32.26±9.12	1.87±0.36
F		34.184	32.389
P		0.000	0.000

3 caspase 3 P53  
n %

Table 3 Comparison of treatment effect of patients with different levels of Caspase 3 and p53 in the observation group

	n	%
Caspase 3 >	25	76.0
	25	44.0
<sup>2</sup>		5.333
P		0.021
P53 >	25	80.0
	25	40.0
<sup>2</sup>		8.333
P		0.004

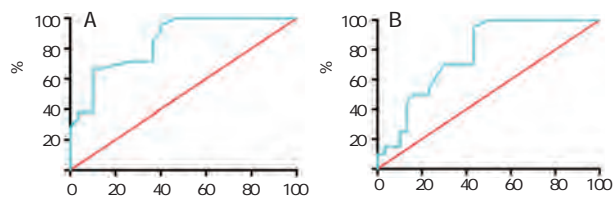
2.4 caspase 3 P53 ROC

caspase 3 P53 ROC

0.771 95%CI 0.642-0.899

14.76 pg/mL

0.835 ng/mL 1



A caspase 3 ROC B P53 ROC

1 caspase 3 P53 ROC  
Figure 1 ROC curve of serum caspase 3 P53 content predicting curative effect

3

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910

11 12

C

1

13 16

caspase 3 P53

caspase 3 P53

P53

pase 3 caspase 3

P53 caspase 3

P53 caspase 3

P53 caspase 3

P53 caspase 3

P53 caspase 3

P53 caspase 3

# RDW COPD

1 2 2 1

RDW COPD

PH 2018 1 2020 1 190 COPD

n=21 4 n=83 PH n=56 PH n=30 PH

SPAP UA BNP RDW

RDW ROC RDW COPD

PH PH FEV1 FEV1/FVC PaO<sub>2</sub> SaO<sub>2</sub> < PH < PH <

PaCO<sub>2</sub> MPA RVOTD RV IL 6 IL 8 TNF hsCRP UA BNP RDW > PH > PH >

P<0.05 LVEF LVESD LVEDD E/A

P>0.05 COPD PH RDW IL 6 TNF

hsCRP BNP COPD PH P<0.05 P>0.05

RDW BNP COPD PH ROC RDW COPD PH

AUC 0.848 95%CI 0.786-0.910 BNP COPD PH AUC 0.856 95%CI 0.785-0.927

RDW COPD COPD

## Expression and correlation of RDW in COPD patients with different degrees of pulmonary hypertension

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**ABSTRACT** Objective To explore the expression and correlation of red blood cell distribution width (RDW) in patients with chronic obstructive pulmonary disease (COPD) complicated with pulmonary hypertension (PH). Methods A total of 190 COPD patients were selected in this hospital from January 2018 to January 2020, and according to the pulmonary artery systolic pressure (SPAP), patients were divided into the normal group (n=83), the mild PH group (n=56), the moderate PH group (n=30), and the severe PH group (n=21). The lung function, blood gas indexes, heart function, serum inflammatory cytokines, uric acid (UA), brain natriuretic peptide (BNP), and RDW were measured and compared in 4 groups. The correlation between RDW and various indicators was analyzed, and the receiver operating characteristic curve (ROC) was used to

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E A  
 LVEF E/A 2  
 2.1  
 1.2.3 FEV1 FEV1/FVC PaO<sub>2</sub> SaO<sub>2</sub>  
 PH < PH < PH < PaCO<sub>2</sub>  
 3 mL 10min - 70 MPA RVOTD RV IL 6 IL 8 TNF hs CRP  
 3000r/min ELISA UA BNP RDW > PH > PH >  
 C 6 IL 6 IL 8 TNF P<0.05  
 hs CRP LVEF LVESD LVEDD E/A  
 P>0.05 1  
 CMIA BNP 2.2 COPD PH RDW  
 RDW COPD  
 1.3 PH RDW IL 6 TNF  
 SPSS 20.0  
 n % <sup>2</sup> -  
 t  
 RDW Pearson  
 Receiver operat  
 ing characteristic ROC RDW COPD  
 HP P<0.05

2 COPD PH RDW  
 Table 2 Correlation analysis of RDW level and various indicators in patients with COPD and PH

	RDW			RDW	
	r	P		r	P
PH	0.492	0.000	LVEDD	0.154	0.113
FEV1	0.170	0.080	LVEDD	0.178	0.067
FEV1/FVC	0.146	0.134	E/A	0.181	0.062
PaO <sub>2</sub>	0.158	0.104	IL 6	0.557	0.000
SaO <sub>2</sub>	0.152	0.118	IL 8	0.165	0.09
PaCO <sub>2</sub>	0.177	0.068	TNF	0.627	0.000
MPA	0.142	0.145	hs CRP	0.735	0.000
RVOTD	0.130	0.182	UA	0.149	0.126
RV	0.163	0.093	BNP	0.546	0.000
LVEF	0.155	0.111	-	-	-

IL 8

COPD

<sup>12</sup> BNP

<sup>13</sup> BNP

COPD

COPD

RDW

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3

COPD

BNP

<sup>8 9</sup>

COPD

COPD

COPD

FEV1/FVC

FEV1

PaCO<sub>2</sub>

PaO<sub>2</sub>

COPD

MPA

RVOTD RDW

COPD

<sup>10</sup> IL 6 COPD

CRP

COPD

<sup>11</sup>

TNF

•

N Osrteoc Crosslaps and TPINP levels in the diagnosis of bone metastases after radiotherapy for nasopharyngeal carcinoma has the largest AUC of 0.872 sensitivity of 82.02% and specificity of 83.78%. The 2 year survival rate of high risk patients with serum N Osrteoc Crosslaps and TPINP was lower than that of low risk patients  $P < 0.05$  . Conclusion Monitoring the levels of N Osrteoc Crosslaps and TPINP can be used to assess bone metastasis in patients with nasopharyngeal carcinoma after radiotherapy which is helpful to provide a reference for the prognosis evaluation of patients.

KEY WORDS N Osrteoc Crosslaps TPINP Nasopharyngeal carcinoma Bone metastasis Tumor markers Prognosis

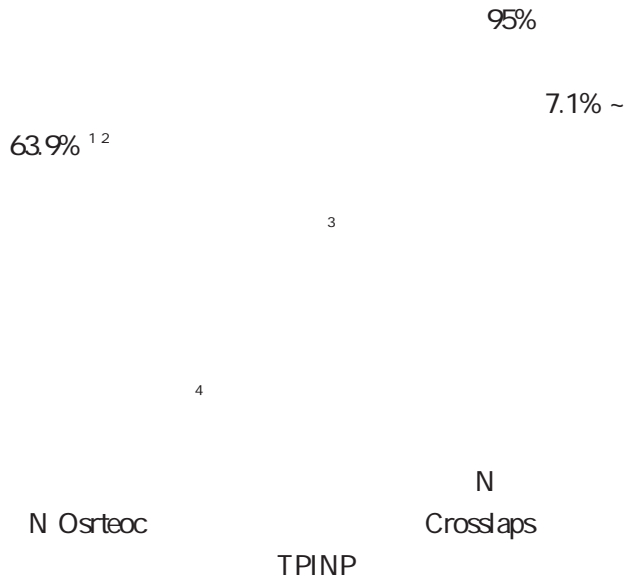


Table 1 Comparison of clinical data between 2 groups

	n %		t <sup>2</sup> / u	P
	n=89	n=37		
50	61 68.54	12 32.43	13.981	<0.001
<50	28 31.46	25 67.57		
	58 65.17	7 18.92	22.384	<0.001
	31 34.83	30 81.08		
	9 10.11	4 10.81	0.244	0.885
	7 7.87	2 5.41		
	73 82.02	31 83.78		
b	13 14.61	12 32.43	3.103	0.002
	29 32.58	17 45.95		
	47 52.81	8 21.62		
	11 12.36	3 8.11	0.145	0.704
	78 87.64	34 91.89		
KPS				
>70	50 56.18	25 67.57	1.407	0.236
70	39 43.82	12 32.43		
Hb				
	82 92.13	34 91.89	0.002	0.963
	7 7.87	3 9.11		
CEA ng/mL	20.61±4.27	13.25±3.71	9.143	<0.001
CYFRA21 1 ng/mL	22.86±6.65	15.63±4.92	5.964	<0.001

Table 2 Comparison of serum N Osrteoc Crosslaps and TPINP levels between 2 groups

	N Osrteoc µg/L	Crosslaps ng/L	TPINP µg/L
n			
89	18.34±4.49	681.16±225.44	87.52±29.01
37	14.13±3.76	430.57±142.39	46.29±15.57
t	5.016	8.866	8.158
P	<0.001	<0.001	<0.001

2.3 Pearson N Osrteoc Crosslaps TPINP

N Osrteoc r=

Table 4 The diagnostic value of serum N Osrteoc Crosslaps and TPINP for bone metastases after radiotherapy for nasopharyngeal carcinoma

	AUC	95%CI	cut off	%	%	P
N Osrteoc	0.744	0.658-0.817	>17.53 µg/L	60.67	81.08	<0.001
Crosslaps	0.827	0.749-0.888	>590.89 ng/mL	69.66	81.08	<0.001
TPINP	0.791	0.710-0.859	>67.79 µg/L	64.04	83.78	<0.001
	0.872	0.800-0.924		82.02	83.78	<0.001

0.881 0.800 Crosslaps r=0.789 0.773 TPINP  
r=0.770 0.760 CEA CYFRA 21 1 P<  
0.05 1

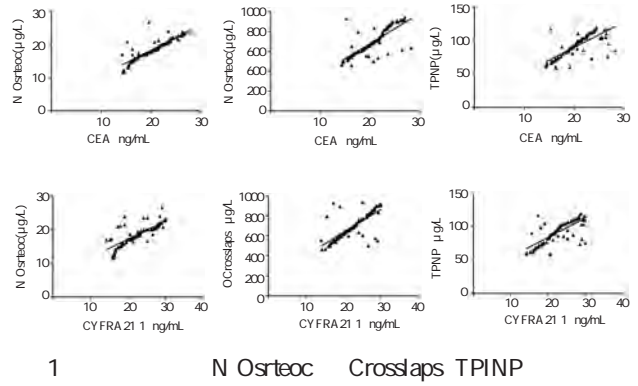


Figure 1 The correlation between serum N Osrteoc, Crosslaps, TPINP and conventional tumor markers in metastasis group

Table 3 Multiple linear regression analysis

	CEA	CYFRA21 1	N Osrteoc	Crosslaps	TPINP	P<0.05
t	11.178	1.052	-	11.537	11.537	<0.001
P	0.826	0.030	0.861	13.304	13.304	<0.001
	0.903	0.022	0.920	14.097	14.097	<0.001
	0.795	0.019	0.811	12.596	12.596	<0.001

Table 3 Multiple linear regression analysis

	t	P
N Osrteoc	11.178	1.052
Crosslaps	11.537	11.537
TPINP	11.537	11.537

2.5 N Osrteoc Crosslaps TPINP

N Osrteoc Crosslaps

TPINP ROC 4

2

4 N Osrteoc Crosslaps TPINP



13 2 847 850

1 EGFL7 MTA1 9 GCs  
J . J .

2020 12 5 616 620

2 J . 10  
2019 25 1 19 22 28

3 2017 9 2 121 126  
CTX N MID Crosslaps J .  
2019 17 2 207 209 230

J . 2018 25 11  
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# Caspase 3

ACI Caspase 3 END  
 2017 7 2019 12 110 ACI ACI  
 60 Caspase 3  
 NIHSS END logistic END ROC  
 Caspase 3 END ACI Caspase 3 17.07±5.34 ng/mL  
 vs. 11.74±3.85 ng/mL ACI Caspase 3  
 ACI END NIHSS 15 hs CRP Caspase 3 END  
 Caspase 3 END END ACI Caspase 3  
 END  
 Caspase 3

## Correlation between serum caspase 3 content and early neurological deterioration in patients with acute cerebral infarction

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**ABSTRACT** Objective To study the correlation between serum caspase 3 content and early neurologic deterioration END in patients with acute cerebral infarction ACI . Methods 110 patients with ACI admitted to the Department of Neurology from July 2017 to December 2019 were selected as ACI group and 60 healthy volunteers in 983 Hospital Joint Logistics Support Force during the same period were selected as the control group. The content of Caspase 3 in serum was detected by enzyme linked immunosorbent assay ELISA END was evaluated by NIHSS and the influencing factor of END was analyzed by logistic regression model and the predictive value of Caspase 3 for END was analyzed by ROC curve. Results serum caspase 3 content in ACI group was higher than that in control group 17.07±5.34 ng/mL vs. 11.74±3.85 ng/mL and serum caspase 3 level in patients with large area infarction and severe infarction was higher than that in patients with small and medium area infarction and mild middle infarction NIHSS score 15 incidence hs CRP and caspase 3 of END patients in ACI group were higher than those of non END patients caspase 3 was the influencing factor of END and had predictive value for END. Conclusion serum caspase 3 content in patients with ACI increases which relates to the severity of the disease and END.

**KEY WORDS** Acute cerebral infarction Caspase 3 Early Neurological Deterioration Prediction Influencing Factors

acute cerebral infarction ACI 1 3 d NIHSS 2

1

early neu 1.4

rological deterioration END ACI

5%~40% ACI END END

ACI 1 2 C hs CRP

ACI END o<sup>-</sup> T<sup>-</sup>

ACI MRI

3 cysteinyl x

aspartate specific proteinase Caspase 3 /6 xMRI x x

10.0 cm<sup>3</sup> >10 cm<sup>3</sup>

NIHSS

15 16! "U-U@ 5 VU@15, Q2 Bg1€

34 5 ACI Cas

ACI Caspase 3

ACI Logistic ROC

Caspase 3 END

1

1.1

2017 7 2019 12

110 ACI ACI ACI 62

48 41~66 51.37±10.25

59 41 40~60

50.12±11.78

P>0.05 ACI

6 48 h

60

1.2 Caspase 3

ACI 5 mL

5 mL

3 000 r/min 10 min

96T

F0037 Caspase 3

1.3 END

7 Nation

al institutes of health stroke scale NIHSS

END 3 d



END  
 END ACI END  
 ACI NIHSS END  
 14 15  
 END ACI  
 NIHSS END  
 Logistic NIHSS END  
 CXCL16  
 HSP70 END CXCL16  
 HSP70  
 16  
 END  
 Caspase 3  
 END Caspase 3  
 END Logistic  
 Caspase 3 ACI  
 Caspase 3  
 END ROC  
 ACI END Caspase 3  
 Caspase 3 END  
 Caspase 3  
 END  
 caspase 3  
 END  
 END  
 ACI Caspase 3  
 END  
 END  
 END  
 END

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 2018 38 1 73 75.

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 2015 48 4 246 257.

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 2020 17 6 1 5

10 rt PA  
 J . 2019 28  
 10 1065 1070.

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 2019 21 8 852 855.

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 70  
 J . 2019 35 24 3803 3807.

2016 9 2019 9 105

Apgar P<0.05

TNF 6 IL 6 fFN NO 2

PGE<sub>2</sub> IGFBP 1 P<

0.05 P<0.05 P>0.05

The clinical value analysis of magnesium sulfate combined with ritodrine hydrochloride in the treatment of preterm premature rupture of membranes

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**ABSTRACT** Objective To analyze the clinical value of magnesium sulfate combined with ritodrine hydrochloride in the treatment of preterm premature rupture of membranes. Method The clinical data of 105 patients with preterm premature rupture of fetal membranes in our hospital from September 2016 to September 2019 were analyzed. According to the random number method they were divided into the study group and the control group. The control group was treated with magnesium sulfate and the study group was treated with magnesium sulfate and ritodrine hydrochloride. The clinical efficacy changes in peripheral blood cytokines vaginal delivery rates and neonatal conditions between two groups were compared. Results The total effective rate and the vaginal delivery rate of the study group were significantly higher than those of the control group and the Apgar score and birth weight of the newborns were significantly higher than those of the control group and the differences between the two groups were statistically significant  $P<0.05$ . After treatment the levels of Tumor Necrosis Factor TNF Interleukin 6 IL 6 Fetal Fibronectin fFN Nitric Oxide NO Prostaglandin E<sub>2</sub> PGE<sub>2</sub> Insulin like Growth Factor Binding Protein IGFBP 1 decreased more than those in the control group. The difference between the two groups was statistically significant  $P<0.05$ . The average uterine contraction inhibition time and the average drug effective time in the study group were shorter than those in the control group and the average gestational age prolonged longer the difference was statistically significant  $P<0.05$ . There was no significant difference in the incidence of adverse reactions between the

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E mail 2500988757@qq.com



2.2  
 2 IL 6 TNF  
 2.1 NO fFN IGFBP 1 PGE<sub>2</sub>

P<0.05 1  
 1 n %  
 Table 1 Comparison of clinical efficacy between 2 groups  
 n %

	n		n	%
	53	28	52.83	24
	45.28	1	1.89	52
	98.11	52	19	36.54
		27	51.92	6
				11.54
				46
				88.46
<sup>2</sup>	-	-	-	-
				3.930
P	-	-	-	-
				<0.05

2.3 P<0.05 2  
 2.4 P<0.05 3  
 Apgar  
 P<0.05 4

2  
 Table 2 Comparison of cytokine changes before and after treatment between the two groups

	IL 6 pg/mL	TNF ng/mL	NO μmol/L	fFN ng/mL	IGFBP 1 ng/mL	PGE <sub>2</sub> pg/mL
	38.89±4.73	3.93±0.62	31.17±3.62	15.17±2.62	68.01±12.12	68.51±7.38
	9.98±1.57	2.43±0.41	24.31±3.17	7.31±2.17	54.03±10.69	43.31±5.03
t	-	42.231	14.691	10.379	16.820	6.298
P	-	<0.001	<0.001	<0.001	<0.001	<0.001
	38.92±4.51	3.84±0.77	31.13±3.26	15.13±4.26	68.13±11.99	68.14±8.00
	15.72±1.33 <sup>a</sup>	3.12±0.49 <sup>a</sup>	28.58±3.27 <sup>a</sup>	9.58±2.27 <sup>a</sup>	60.11±9.71 <sup>a</sup>	52.07±5.35 <sup>a</sup>
t	-	35.580	5.689	3.982	8.291	3.748
P	-	<0.001	<0.001	<0.001	<0.001	<0.001

<sup>a</sup>P<0.05

3  
 Table 3 Comparison of clinical indicators between 2 group after treatment  
 11.54% 3.77% 3  
<sup>2</sup>=2167 P=0.141

	n	h	d	h
	53	217±0.83	17.26±5.81	20.71±6.55
	52	4.43±1.37	10.33±5.19	29.02±6.63
t	-	10.246	6.442	6.461
P	-	<0.001	<0.001	<0.001

4 n %  
 Table 4 Comparison of pregnancy outcomes and condition of newborn between the two 2 groups n %

	n	kg	Apgar
	53	49	92.45
	4	7.54	3.35±1.02
	9.36±2.27	52	40
	76.92	12	23.08
			2.34±1.16
			7.63±2.14
t/ <sup>2</sup>	-	4.901	4.740
P	-	0.027	<0.001
			<0.001

2.5

1 1 12 IL 6 TNF

IL 6 TNF

				5	.	+
		13	fFN IGFBP 1		J .	2019 6 4
				102	103.	
		14	PGE <sub>2</sub>	6	.	J .
					2018 41 05 103 104.	
PGE <sub>2</sub>				7	.	J .
			PGE <sub>2</sub>		2016 14 23 110 111.	
		15 16	NO	8	Doret M Kayem G. Tocolysis for preterm labor without pre	
			PGE <sub>2</sub>		mature preterm rupture of membranes J . J De Gynecol Ob	
			PGE <sub>2</sub>		stetrique Et Biologie De La Rep 2016 44 9 89 92	
		17		9	.	J .
NO					2017 45 46 46.	
		18		10	Juliana SE Renato Augusto Moreira de Sá Paulo Roberto	
					Nassar de Carvalho et al. Neonatal outcome in women with	
					preterm premature rupture of membranes PPRM between	
					18 and 26 weeks J . J Mat Fetal Med 2016 29 7 1108	
					1112	
				11	.	J .
					2018 43 12 69 75.	
				12	.	J .
					2019 35 7 498 501.	
				13	.	J .
					2018 47 3 100 104.	
				14	Su AK Park KH Lee SM. Non Invasive Prediction of Histo	
					logic Chorioamnionitis in Women with Preterm Premature	
					Rupture of Membranes J . 2016 57 2 461 468.	
				15	.	J .
					2019 28	
					6 13 14.	
				16	.	J .
					2018 27 11 33 39.	
				17	.	J .
					2017 14 26 146 148.	
				18	Sentilhes L Sénat MV Ancel PY et al. Prevention of	
					spontaneous preterm birth excluding preterm premature rup	
					ture of membranes Guidelines for clinical practice Text of	
					the Guidelines short text J . 2016 45 10 1446 1456.	
				19	.	J .
					2019 16 2 157 159.	
				20	.	J .
					NO IL 6 PGE2	
					2017 32 10 2150 2152.	
					J .	
1					.	
					2017 20 1 40 42	
2					.	
					2019 11 4	
					256 262 294.	
3					.	
					2018 34 12 2422 2423	
4					.	
					2018 31 3 111 112	

# AFP GT ApoA1

A1 ApoA1

2013 1 2017 12 180

AFP GT ApoA1

ApoA1

7d

P<0.05 ROC

AUC 0.695

P<0.05

P<0.05

P<0.05

ApoA1

P<0.05

A1

## Changes of serum AFP GT ApoA1 levels before and after operation in patients with liver cancer and their clinical significance

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**ABSTRACT** Objective To explore the changes and clinical significance of serum alpha fetoprotein (AFP), glutamyl transpeptidase (GT) and apolipoprotein A1 (ApoA1) in patients with liver cancer before and after surgery. Methods The clinical data of 180 patients undergoing liver cancer surgery from January 2013 to December 2017 were retrospectively analyzed. The AFP, GT and ApoA1 before and after surgery were measured and evaluated. The relationship between levels of AFP, GT and ApoA1 before surgery and clinicopathological features was analyzed. The survival time of patients was recorded at follow up. The relationship between AFP, GT and ApoA1 before and after surgery and liver cancer recurrence and patient survival was observed. Results At 7d after surgery, the AFP and GT levels were lower than those before surgery, while the ApoA1 was higher than that before surgery ( $P < 0.05$ ). ROC curve showed that AFP and GT before and after surgery and ApoA1 after surgery were effective indicators for the diagnosis of liver cancer recurrence, and the AUC of AFP before surgery in the diagnosis of liver cancer recurrence was 0.695. The proportions of patients with high AFP and high GT before liver cancer surgery in multiple tumors, portal

vein thrombosis and poor tissue differentiation were higher than those of patients with low values  $P < 0.05$  and the proportions of patients with high ApoA 1 before liver cancer surgery in multiple tumors and poor tissue differentiation were lower than those of patients with low value  $P < 0.05$  and the proportion of patients with high AFP before liver cancer surgery in vascular involvement was higher than that of patients with low value  $P < 0.05$  and the proportion of patients with high GT before liver cancer surgery in lymph node metastasis was higher than that of patients with low value  $P < 0.05$ . The survival rates of patients with high AFP and high GT after surgery were lower than those of patients with low values and the survival times were shorter than those of patients with low values and the survival rate of patients with high ApoA 1 after surgery was higher than that of patients with low value and the survival time was longer than that of patients with low value  $P < 0.05$ . Conclusion AFP and GT in liver cancer patients after surgery decreased and ApoA 1 increased. Monitoring the levels of AFP and GT before and after surgery and their changing trends can provide references for predicting the prognosis of patients.

KEY WORDS Alpha fetoprotein glutamyltranspeptidase Apolipoprotein A 1

140 40  
 5 26 21  
 50% 5 60%~70% 159 27  
 1 114

2

Alpha fetoprotein AFP  
 3 AFP  
 glutamyl transpeptidase GT 1.1.2 /  
 AFP  
 4 GT  
 ApoA 1  
 A 1 Apolipoprotein A 1 ApoA 1  
 5 ApoA 1 CTK 132C  
 6 ApoA 1 AU480

1.2  
 AFP GT ApoA 1  
 AFP GT ApoA 1  
 1 3 mL 3 500 r/min 7 d  
 1.1 - 20 5 min  
 1.1.1 AFP ApoA 1 GT  
 1.3 SPSS 19.0  
 180 - t  
 144 36 40~78  
 53.27±11.84 156 n % 2 Kaplan Meier  
 52 21 log rank

P<0.05  
 2  
 2.1  
 AFP GT ApoA1  
 7 d  
 AFP  
 ApoA1  
 P<0.05  
 1  
 1  
 AFP GT ApoA1

2.2  
 AFP GT ApoA1  
 ROC  
 ApoA1  
 AUC  
 0.695  
 AFP  
 P<0.05  
 1 2  
 AUC

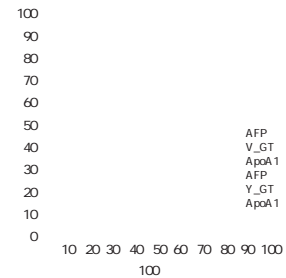


Table 1 Changes of levels of AFP GT and ApoA1 before and after liver cancer surgery

	n	AFP μg/L	GT U/L	ApoA1 g/L
	180	313.17±87.93	72.25±4.97	0.79±0.32
7 d	180	23.71±5.04	58.53±10.60	0.89±0.22
t	-	44.094	15.723	- 3.455
P	-	<0.0001	<0.0001	<0.0001

Figure 1 ROC curves of AFP, GT and ApoA1 before and after surgery in the diagnosis of recurrence after liver cancer surgery

Table 2 Relationship between AFP GT and ApoA1 before and after surgery and liver cancer recurrence

	AUC	%	%	95%CI	P
AFP	0.695	327.35	0.628	0.610-0.771	<0.001
GT	0.639	75.49	0.490	0.552-0.720	0.007
ApoA1	0.550	0.78	0.549	0.462-0.635	0.334
AFP	0.617	27.80	0.392	0.529-0.699	0.024
GT	0.639	62.87	0.510	0.564-0.731	0.003
ApoA1	0.610	0.710	0.431	0.523-0.694	0.037

2.3 AFP GT ApoA1 78.33% 18 121 67.22%

AFP GT  
 ApoA1  
 P<0.05  
 AFP  
 P<0.05  
 GT  
 P<0.05 3

3  
 AFP  
 7 8  
 AFP  
 ApoA1  
 9  
 HBV  
 TNM  
 GT  
 10 11

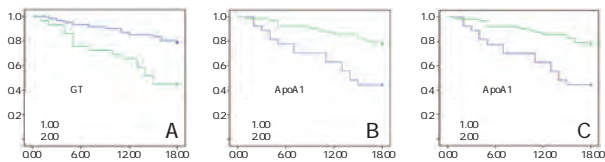
2.4 AFP GT ApoA1  
 AFP GT  
 P<0.05  
 ApoA1  
 P<0.05 4 2  
 2.5  
 180 12 55 1  
 30.56% 18 68 37.78%  
 180 12 141 1  
 AFP  
 AFP

3 AFP GT ApoA1 n %  
 Table 3 Relationship between levels of AFP GT and ApoA1 before surgery and clinicopathological features n %

	n	AFP µg/L		GT U/L		ApoA1 g/L	
		n=77	n=103	n=49	n=131	n=96	n=84
	40	26 33.77	14 13.59	24 48.98	16 12.21	14 14.58	26 30.95
	140	51 66.23	89 86.41	25 51.02	115 87.79	82 85.42	58 69.05
<sup>2</sup> P		10.375 0.001		27.890 <0.0001		6.945 0.008	
	26	16 20.78	10 9.71	18 36.73	8 6.11	12 12.50	14 16.67
	154	61 79.22	93 90.29	31 63.27	123 93.89	84 87.50	70 83.33
<sup>2</sup> P		4.370 0.037		27.069 <0.0001		0.629 0.428	
	21	16 20.78	5 4.85	12 24.49	9 6.87	4 4.17	17 20.24
	159	61 79.22	98 95.15	37 75.51	122 93.13	92 95.83	67 79.76
<sup>2</sup> P		10.843 0.001		10.743 0.001		11.228 0.001	
	27	12 15.58	15 14.56	15 55.56	12 9.16	11 11.46	16 19.05
	153	65 84.42	88 85.44	34 22.22	119 90.84	85 88.54	68 80.95
<sup>2</sup> P		0.036 0.849		12.871 <0.0001		2.024 0.155	
	114	63 81.82	51 49.51	30 61.22	84 64.12	64 66.67	50 59.52
	66	14 18.18	52 50.49	19 38.78	47 35.88	32 33.33	34 40.48
<sup>2</sup> P		19.800 <0.0001		0.129 0.720		0.984 0.321	

4 AFP GT ApoA1  
 Table 4 Relationship between AFP, GT and ApoA1 after surgery and patient survival time

	AFP		GT		ApoA1	
	30.00	12/39	10.00	4.25 18.00	44.64	25/56
	78.57	109/141	18.00	18.00 18.00	77.42	96/124
<sup>2</sup> Z	30.026	26.497	18.809	11.727	26.814	11.605
P	<0.0001	<0.0001	<0.0001	0.001	<0.0001	0.001



A AFP B GT C ApoA1  
 2 AFP GT ApoA1

Figure 2 Survival curves of patients with postoperative high and low AFP GT ApoA1

12

AFP mRNA

13

AFP

AFP

AFP

15

A

ApoA1

1526

# OSAHS

OSAHS  
65 OSAHS

AHI 22 23 20 3

CPAP  
18 IL 18

TNF 1 ET 1 IMT HDL ch

LDL ch SaO<sub>2</sub> SaO<sub>2</sub>

AHI IMT IL 18 TNF ET 1 HDL ch LDL ch

P>0.05 OSAHS SaO<sub>2</sub> SaO<sub>2</sub> AHI

IMT P<0.05 OSAHS SaO<sub>2</sub>

AHI P<0.05 IMT LDL ch HDL ch

P>0.05 OSAHS SaO<sub>2</sub> SaO<sub>2</sub>

AHI IMT P<0.05 OSAHS IMT

OSAHS IL 18 TNF ET 1

## Correlation analysis between related inflammatory factors in patients with OSAHS and atherosclerosis

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**ABSTRACT** Objective To analyze the expression of related inflammatory factors in patients with obstructive sleep apnea hypopnea syndrome (OSAHS) and the correlation with atherosclerosis. Method 65 patients with OSAHS admitted to our department of respiratory medicine were selected and were divided into the mild group (22 cases), the moderate group (23 cases), and the severe (20 cases) group according to the sleep apnea hypopnea index (AHI). The patients were treated with continuous positive airway pressure (CPAP) and healthy subjects (25 patients) who underwent routine physical examination at the same time were selected as the control group and interleukin

glyceride levels in the four groups  $P > 0.05$ . In patients with OSAHS the average  $SaO_2$  and the lowest  $SaO_2$  decreased gradually with the severity of the disease and AHI carotid IMT and the above inflammatory factors gradually increased and the differences in the four groups were statistically significant  $P < 0.05$ . After treatment the lowest  $SaO_2$  in patients with OSAHS significantly increased AHI carotid IMT and the above inflammatory factors significantly decreased  $P < 0.05$ . There was no significant difference between the three groups in the carotid IMT LDL ch HDL ch and triglycerides  $P > 0.05$ . Correlation analysis showed that the levels of above inflammatory factors in OSAHS patients were negatively correlated with mean  $SaO_2$  and lowest  $SaO_2$  and positively correlated with AHI and carotid IMT  $P < 0.05$ . Conclusion In patients with OSAHS the carotid IMT is thickened and the level of inflammatory factors is positively correlated with the severity of the disease.

KEY WORDS OSAHS Atherosclerosis IL 18 TNF ET 1

Obstruc  
tive Sleep Apnea Hypopnea Syndrome OSAHS

25  
14 11

1 OSAHS 43.07±9.51  
2  $P > 0.05$   
1.2  
18 Interleukin 18 IL 18  
Tumor Necrosis Factor TNF 1 En  
dothelin 1 ET 1  
3 OSAHS  
Continuous Positive Airway Pressure  
CPAP IL 18 TNF ET 1  
Intima Media  
Thickness IMT High Den  
sity Lipoprotein HDL ch  
Low Density Lipoprotein LDL ch  
OSAHS  
CAPA  
CAPA 5  
1 5-8 h 8-15  
1.1 cm H<sub>2</sub>O 3  
SaO<sub>2</sub>  
2 65  
CPAP  
25-67 43.72±9.55 OSAHS 1.5 cm  
35 30  
IMT 3  
Apnea Hypopnea Index AHI 4  
5 mL  
PSG OSAHS IL 18 TNF  
ET 1  
HDL ch LDL ch

1.3

P<0.05

6  
 2  
 SaO<sub>2</sub> 4%  
 30% >10s  
 3% >10s  
 50%  
 HDL ch  
 OSAHS AHI  
 IMT  
 SaO<sub>2</sub> SaO<sub>2</sub>  
 1.4  
 SPSS 18.00  
 P<0.05 LDL ch  
 P>0.05 OSAHS  
 tF n % SaO<sub>2</sub> SaO<sub>2</sub> AHI IMT  
 2  
 Pearson P<0.05 1  
 1 -

Table 1 Comparison of sleep and atherosclerosis indicators in each group

	n=25	OSAHS			F	P
		1	2	3		
SaO <sub>2</sub> %	95.56±2.23	93.10±2.52 <sup>a</sup>	88.79±5.43 <sup>ab</sup>	83.26±2.62 <sup>abc</sup>	52.91	<0.001
SaO <sub>2</sub> %	92.13±3.14	86.13±5.15 <sup>a</sup>	82.17±5.51 <sup>ab</sup>	71.64±5.46 <sup>abc</sup>	68.81	<0.001
AHI /h	2.13±1.01	10.31±2.54 <sup>a</sup>	22.64±3.78 <sup>ab</sup>	40.61±5.35 <sup>abc</sup>	515.71	<0.001
LDL ch mmol/L	2.86±0.83	2.90±0.82	2.94±0.77	2.96±0.85	0.07	0.977
HDL ch mmol/L	1.23±0.24	1.03±0.15 <sup>a</sup>	1.07±0.18 <sup>a</sup>	0.99±0.22 <sup>a</sup>	6.39	<0.001
mmol/L	1.03±0.51	2.21±2.04	1.54±1.58	2.01±1.85	2.60	0.058
IMT mm	0.66±0.09	0.74±0.12 <sup>a</sup>	0.87±0.18 <sup>b</sup>	1.06±0.15 <sup>abc</sup>	34.84	<0.001

<sup>a</sup>P<0.05

<sup>b</sup>P<0.05

<sup>c</sup>P<0.05

2.2

P<0.05

IL 18 TNF ET 1 2  
 2 - )  
 Table 2 Comparison of levels of inflammatory factors in each group - )

	n=25	OSAHS			F	P
		1	2	3		
IL 18 ng/L	249.86±76.23	352.67±76.13 <sup>a</sup>	601.02±84.54 <sup>ab</sup>	797.61±112.34 <sup>abc</sup>	175.58	<0.001
TNF ng/L	12.33±3.39	17.64±3.01 <sup>a</sup>	23.74±2.98 <sup>ab</sup>	36.07±3.37 <sup>abc</sup>	221.63	<0.001
ET 1 ng/L	0.61±0.13	0.82±0.14 <sup>a</sup>	1.05±0.22 <sup>ab</sup>	1.67±0.31 <sup>abc</sup>	104.70	<0.001

<sup>a</sup>P<0.05

<sup>b</sup>P<0.05

<sup>c</sup>P<0.05

2.3

OSAHS

IMT

3

SaO<sub>2</sub> OSAHS  
 AHI IL 18 TNF ET 1 P<0.05  
 IMT HDL ch LDL ch IMT  
 P>0.05 3

2.4

OSAHS

Pearson OSAHS OSAHS OSAHS OSAHS IMT  
 IL 18 TNF ET 1 SaO<sub>2</sub> SaO<sub>2</sub> OSAHS OSAHS  
 AHI IMT P< OSAHS  
 0.05 4 8

		t	
	SaO <sub>2</sub> %	90.87±7.94	97.86±8.77
		74.15±3.88	87.85±7.13
AHI	/h	30.31±3.93	7.52±3.04
		36.23±5.19	8.34±2.57
		47.64±6.56	9.20±3.14
	IMT		
	mm	0.93±0.12	0.90±0.07
		0.96±0.11	0.91±0.09
		0.98±0.14	0.91±0.10
IL	18 ng/L	356.98±73.63	272.48±68.10
		612.48±82.46	300.47±75.46
		754.23±120.45	339.35±88.42
TNF	ng/L	19.31±3.06	13.57±2.84
		25.77±2.45	14.64±3.26
		35.28±3.88	16.43±3.35
ET	1 ng/L	0.85±0.17	0.68±0.15
		1.47±0.26	0.71±0.16
		1.72±0.29	0.79±0.20
LDL ch	mmol/L	2.28±0.74	2.25±0.77
		2.56±0.85	2.33±0.72
		2.63±0.92	2.45±0.78
HDL ch	mmol/L	1.05±0.27	1.03±0.26
		1.06±0.25	1.03±0.27
		1.02±0.24	0.99±0.25
	mol/L	2.10±1.32	1.87±1.21
		2.16±1.38	1.75±1.17
		2.11±1.34	1.78±1.16

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ApoA1

AFP GT  
AFP GT  
ApoA1

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## Th1 Th2 Th17 Treg

T 17 Th17 T 98 Treg 17 IL 17 TNF 53 T 1 Th1 2017 2 Th2 2019 2  
 Th1 Th2 Th17 Treg IFN SWI Th1  
 Th2 Th17 Treg 1 Th1 Th2 Th17  
 Treg IL 17 TNF IL 4 IFN Th2 Treg  
 P<0.05 Th2 Treg P<0.05  
 Th1 Th17 IL 17 TNF IL 4 IFN P<  
 Th1 Th17 IL 17 TNF IL 4 IFN P<  
 0.05 Th2 Treg P<  
 0.05 Th1 Th17  
 P<0.05 logistics Th1 Th2 Th17 Treg  
 P<0.05 Th1 Th2 Th17 Treg  
 Th1 Th2 Th17 Treg

### Changes and significance of peripheral blood Th1 Th2 Th17 and Treg cells in patients with massive cerebral infarction

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**ABSTRACT** Objective To observe the changes and significance of peripheral blood Th1 Th2 Th17 and Treg cells in patients with massive cerebral infarction. Methods 98 patients with massive cerebral infarction admitted to the hospital between February 2017 and February 2019 were selected as the observation group and 53 normal people who complete physical examination during the same period were selected as the control group. The levels of Th1 Th2 Th17 Treg cells interleukin 17 IL 17 and tumor necrosis factor TNF were measured and compared. Patients in the observation group completed susceptibility weighted imaging SWI . The correlation between the infarct size number of infarcts and Th1 Th2 Th17 Treg cells was analyzed. After 1 month of routine treatment the levels of Th1 Th2 Th17 Treg cells IL 17 TNF IL 4 and IFN in the observation group before and after treatment were compared. Results Th2 and Treg in the observation group were significantly lower than those in the control group with statistical significance P<0.05 . Besides Th2 and Treg in observation group before treatment were significantly lower than those after treatment with statistical significance P<0.05 . Th1 Th17 IL 17 TNF IL 4 and IFN were

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significantly higher than those in the control group with statistical significance  $P < 0.05$ . Besides the above indicators in the observation group before treatment were significantly higher than those after treatment with statistical significance  $P < 0.05$ . The levels of Th2 and Treg cells in peripheral blood were negatively correlated with the number volume and depth of cerebral infarction lesions  $P < 0.05$ . However the levels of Th1 and Th17 cells in peripheral blood were positively correlated with the number volume and depth of cerebral infarction lesions  $P < 0.05$ . Logistics regression analysis showed that the levels of Th1 Th2 Th17 and Treg cells in peripheral blood were significantly correlated with the diagnostic results of massive cerebral infarction  $P < 0.05$ . Conclusion The development of large area cerebral infarction is closely related to the levels of Th1 Th2 Th17 and Treg cells in the peripheral blood of patients. The levels of Th1 Th2 Th17 and Treg cells can be used as independent risk factors for the onset of large area cerebral infarction.

KEY WORDS Massive cerebral infarction Th1 Th2 Th17 Treg

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mL Th1 Th2 Th17 Treg  
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Treg BioSystems  
A15  
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17 interleukin17 IL 17  
T 1 T helpercell 1 Th1 a tumor necrosis factor TNF 14  
T 2 T helpercell 2 Th2 T 17 interfeuron  
T helpercell 17 Th17 T regulato IFN  
ry T cells Treg  
1.2.2 SWI  
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98 3D MIP SWI  
53 5 T1WI T2WI SWI  
53 45 27~79 48.89± 1 2  
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Spearman logistics  
P<0.05

2.2 P<0.05 1  
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 P<0.05  
 Th1 Th17 IL 17 TNF IL 4 Th1 Th17 IL 17 TNF IL 4 IFN  
 IFN P<0.05  
 P<0.05  
 2  
 1 Th1 Th2 Th17 Treg IL 17 TNF IL 4 IFN -

Table 1 Comparison of Th1 Th2 Th17 Treg cells IL 17 TNF IL 4 and IFN between 2 groups

	n	Th1 %	Th2 %	Th17 %	Treg %	IL 17 ng/L	TNF ng/L	IFN ng/L	IL 4 ng/L
	98	19.14±5.53	1.39±0.32	18.47±3.15	2.24±0.41	35.79±0.2	68.79±11.02	18.79±3.07	15.79±3.22
	53	11.59±3.47	2.68±0.76	5.89±1.21	4.19±0.57	24.43±0.87	44.43±10.87	4.43±0.87	7.43±1.87
t		8.850	6.629	15.639	7.541	8.805	12.444	13.172	9.805
P		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

Table 2 Comparison of Th1 Th2 Th17 Treg cells IL 17 TNF IL 4 and IFN in the observation group before and after treatment

	n	Th1 %	Th2 %	Th17 %	Treg %	IL 17 ng/L	TNF ng/L	IFN ng/L	IL 4 ng/L
	98	19.14±5.53	1.39±0.32	18.47±3.15	2.24±0.41	35.79±0.2	68.79±11.02	18.79±3.07	15.79±3.22
	98	13.54±3.31	2.14±0.71	8.81±2.12	3.48±0.34	26.44±0.88	48.45±9.83	8.47±0.87	9.42±1.87
t		6.823	5.629	11.647	5.514	7.812	10.245	11.873	7.834
P		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

2.3 Th1 Th2 Th17 Treg  
 SWI 98  
 357  
 208 149 10  
 cm³< 15 cm³ 37 15 cm³<  
 <25 cm³ 32 >25 cm³ 29 Spear  
 man Th2  
 Treg

Table 3 Correlation analysis of peripheral blood Th1 Th2 Th17 and Treg in observation group

	r	P	r	P	r	P
Th1	0.337	<0.05	0.313	<0.05	0.314	<0.05
Th2	-0.515	<0.05	-0.412	<0.05	-0.506	<0.05
Th17	0.531	<0.05	0.427	<0.05	0.514	<0.05
Treg	-0.441	<0.05	-0.353	<0.05	-0.340	<0.05

P<0.05  
 Th1 Th17  
 P<0.05 3

Table 4 Correlation analysis between the levels of peripheral blood Th1 Th2 Th17 and Treg cells and the diagnosis results of massive cerebral infarction

	SE	Wald/²	OR	95%CI	P	
Th1	0.052	0.023	4.693	1.042	1.012-1.104	0.028
Th2	-0.173	0.054	4.232	1.169	0.753-0.993	0.037
Th17	0.087	0.036	4.956	1.188	1.013-1.174	0.024
Treg	-0.113	0.048	4.754	1.096	1.021-1.195	0.013

2.4 Th1 Th2 Th17 Treg  
 0 1  
 Th1 Th2 Th17 Treg  
 logistics Th1 Th2  
 Th17 Treg  
 P<0.05 4

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6 7

T  
89

T Th T  
cytotoxic T cells Tc Treg

10

Th1 Th2 Th17

Treg

Th1 Th2 Th17

Treg IL 17 TNF

11

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CD4+T NKT CD8+T  
CD4+T Th Th1 Th2

Th1 CD4+T

IL 2 IFN Th2

B IL 4 IL 10  
Th17 Treg T

Th17 IL 17 TNF

Th1 Th17

IFN IL 17

Th17/Treg Th17

Treg

Treg

CD8+T CD4+T TNF  
T

13

Treg

Treg  
Th1 Th17  
Th2 Treg  
Spearman  
Th1/Th2 Th17/

Treg IFN IL 4 IL 17  
TNF 14 15

Th1 Th2 Th17 Treg Th1  
Th2 Th17 Treg

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J . 2020 12  
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oxLDL  
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%\* # " ' # "

# IL 6 sICAM 1

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IL 6 sICAM 1 NP  
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40 IL 6

sICAM 1 46

37 IL 6 sICAM 1 IL 6 sICAM 1 NP

ROC AUC

IL 6 sICAM 1 P<0.05 IL 6 sICAM 1 AUC

0.952 0.796 IL 6 AUC sICAM 1 P<0.05

IL 6 sICAM 1

P<0.05 IL 6 sICAM 1 NP

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## Application of serum IL 6 and sICAM 1 in the differential diagnosis of neonatal pneumonia bacterial infection and evaluation of curative effect

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**ABSTRACT** Objective To explore the application of serum interleukin 6 IL 6 and soluble inter cellular adhesion molecules 1 sICAM 1 in the differential diagnosis of neonatal pneumonia NP bacterial infection and evaluation of curative effect. Methods A total of 83 children with NP admitted to our hospital from January 2019 to December 2019 were selected as the pneumonia group and 40 normal newborns who had a normal physical examination in the pediatric care department of our hospital during the same period were selected as the control group. The venous blood of children in the control group and the pneumonia group was collected when they were admitted to the hospital and the levels of serum IL 6 and sICAM 1 were detected. The sputum culture test was performed on the children in the pneumonia group. According to the type of infec

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tious pathogens they were divided into 46 cases of the bacterial infection group and 37 cases of the non bacterial infection group. The level of IL 6 and sICAM 1 were detected when the newborns in the bacterial infection group recovered. The diagnostic efficacy of serum IL 6 and sICAM 1 on NP bacterial infections was analyzed using receiver operating characteristic curve ROC and area under the curve AUC was calculated. Results The levels of serum IL 6 and sICAM 1 in the bacterial infection group were significantly higher than those in the non bacterial infection group the difference was statistically significant  $P < 0.05$  . AUC values of serum IL 6 and sICAM 1 were 0.952 and 0.796 respectively which had high diagnostic value for the disease. The AUC of IL 6 was significantly higher than that of sICAM 1  $P < 0.05$  . The levels of serum IL 6 and sICAM 1 in the bacterial infection group were significantly higher than those in the control group during the acute phase which were significantly decreased during the recovery phase after treatment. The difference was statistically significant  $P < 0.05$  . Conclusion Serum IL 6 and sICAM 1 have important clinical detection significance in the diagnosis of NP bacterial infection.

KEY WORDS Neonatal pneumonia Bacterial infection Interleukin 6 Soluble intercellular adhesion molecule 1 Curative effect evaluation

neonatal pneumonia NP

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IL 6

6 Interleukin 6

1 sICAM 1



IL 6

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<sup>14</sup> ICAM 1

ICAM 1

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15

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IL 6

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16

17

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ROC

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NP

IL 6

IL 6 sICAM 1

NP

NP

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# FIB FDP D D TAT

FIB FDP D D TAT

2018 2 2020 1

DVT 46 DVT 46

DVT DVT 1 3d FIB FDP D D

PT APTT TAT

ROC

FIB FDP D D TAT DVT > DVT >

P<0.05 DVT 1 3d FIB FDP D D TAT DVT P<0.05 D D

FIB FDP D D TAT P<0.05 D D

73.17% 100.00% FDP TAT FIB

97.56% 100.00% FIB FDP D D TAT

DVT DVT D D

DVT

D

## Relationship between serum FIB FDP D D and TAT levels and thrombosis in patients with traumatic limbs fractures

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**ABSTRACT** Objective To explore the relationship between serum fibrinogen FIB fibrin degradation products FDP D dimer D D and thrombin anti-thrombin complex TAT levels and thrombosis in patients with traumatic limbs fractures to provide reference for preventing thrombosis. Methods A retrospective collection was performed on the 46 patients with traumatic limbs fractures and deep vein thrombosis DVT who were admitted from February 2018 to January 2020 DVT group while another 46 patients with traumatic limbs fractures and without DVT group.

thrombosis was analyzed by receiver operating characteristic (ROC) curves. Results: FIB, FDP, D-D and TAT were the highest in the DVT group, followed by the non-DVT group, and were the lowest in the control group ( $P < 0.05$ ). Before surgery, 1 d and 3 d after surgery, FIB, FDP, D-D and TAT in the DVT group were higher than those in the non-DVT group ( $P < 0.05$ ). Preoperative FIB, FDP, D-D and TAT were all influencing factors of thrombosis in patients with traumatic limb fractures ( $P < 0.05$ ). The efficiency of D-D alone was the highest in predicting thrombosis, and the sensitivity and specificity were 73.17% and 100.00%, followed by FDP, TAT and FIB. The combined predictive efficiency of the four was higher than that of alone, and the sensitivity and specificity were 97.56% and 100.00%, respectively. Conclusion: The FIB, FDP, D-D and TAT in patients with traumatic limb fractures are higher than those in normal people. The concentrations of the above indicators in DVT patients are higher than those in non-DVT patients. The efficiency of D-D alone is the highest in predicting thrombosis of traumatic limb fractures. The combination of the four can improve early detection rate of DVT.

KEY WORDS Traumatic limb fracture, Fibrinogen, D-dimer, Fibrin degradation products, Thrombin-anti-thrombin complex

		4	DVT	30	16	
1		48.79±11.51	BMI	27	22.97±3.45	kg/m <sup>2</sup>
				31	12	
		3			46	
	Deep venous thrombosis (DVT)			28	18	48.95±
2		DVT	10.75	BMI	22.87±3.25	kg/m <sup>2</sup>
				BMI		P>0.05
	Wells					
		3				
	D-Dimer (D-D)			>16		
		45		DVT	DVT	1~
			3d		DVT	
	DVT					6
	DVT					
1						
1.1						
	2018	2	2020	1		
			46		DVT	
	1	1				
	DVT	46	DVT	DVT	29	
	17		49.51±10.43			
	Boby mass index (BMI)		22.71±3.11		kg/m <sup>2</sup>	
		26		20		
		30		12		

Activated partial prothrombin time n %<sup>2</sup>  
 APTT D D Logistic Receiver operating character  
 Thrombin an  
 ti thrombin compounds TAT istic curve ROC  
 BECKMAN COULTER ACL Advance P<0.05  
 1.3 2.1 3  
 SPSS 20.0 3 PT APTT P>  
 - t LSD t 0.05 FIB FDP D D TAT DVT > DVT  
 P<0.05

		FIB g/L	FDP µg/L	D D µg/L	PT s	APTT s	TAT µg/L	
DVT	46	2.61±0.53	3.79±1.15	229.63±50.14	12.14±2.14	26.31±4.45	1.45±0.36	
DVT	46	3.45±0.51 <sup>a</sup>	19.12±4.77 <sup>a</sup>	379.52±66.75 <sup>a</sup>	12.43±1.98	26.44±4.15	3.15±0.65 <sup>a</sup>	
DVT	46	3.99±0.43 <sup>ab</sup>	35.75±10.23 <sup>ab</sup>	571.43±115.58 <sup>ab</sup>	12.67±2.33	26.57±3.78	4.87±1.61 <sup>ab</sup>	
F		91.937	273.904	199.271	0.698	0.045	128.341	
P		<0.001	<0.001	<0.001	0.499	0.956	<0.001	4

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Table 3 Analysis of the influence of preoperative coagulation and fibrinolysis parameters on thrombosis in traumatic limb fractures

	S.E.	Wald/ <sup>2</sup>	OR	95%CI	P
FIB	0.655	0.236	7.703	1.925 1212~3.057	0.006
FDP	0.711	0.267	7.091	2.036 1.206~3.436	0.008
D D	0.997	0.165	36.511	2.710 1.961~3.745	<0.001
PT	0.052	0.236	0.049	1.053 0.663~1.673	0.826
APTT	0.067	0.177	0.143	1.069 0.756~1.513	0.705
TAT	0.985	0.103	91.453	2.678 2.188~3.277	<0.001

FIB 73.17% 100.00% FDP TAT  
4 1

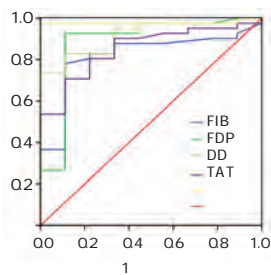
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DVT  
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DVT DVT  
FIB FDP D D TAT  
DVT  
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DVT  
DVT

4

Table 4 Value analysis of preoperative coagulation and fibrinolysis parameters in predicting thrombosis in traumatic limb fractures

	cut off	AUC	95%CI		%	%	P
FIB	>3.55	0.820	0.685-0.914	0.669	78.05	88.89	<0.001
FDP	>19.88	0.885	0.763-0.958	0.816	92.68	88.89	<0.001
D D	>384.10	0.916	0.802-0.976	0.732	73.17	100.00	<0.001
TAT	>3.61	0.852	0.723-0.937	0.596	70.73	88.89	<0.001
	-	0.978	0.891-0.999	0.976	97.56	100.00	<0.001



1

Figure 1 Value analysis of preoperative coagulation and fibrinolysis parameters in predicting thrombosis in traumatic limb fractures

Korte <sup>9</sup>  
FIB FDP  
DVT  
D D  
10 TAT  
TAT DVT  
Negreva <sup>13</sup>  
DVT  
DVT  
D D  
DVT  
DVT  
D D  
FIB FDP D D TAT  
DVT  
FIB FDP D D  
1543

,LDHLRP

LDHLRP (

• •

# KL 6 LDH

LRP DD 137 n=45

6 KL 6 LDH 2014 1 2016 6 n=71

LDH CRP D DD n=66

KL 6 LDH CRP DD ! /

/ WÂ' ÀP 1P 1P

were higher than those in the survival group P<0.05 . Conclusion Serum KL 6 and LDH can be used as biomarkers of connective tissue disease complicated with interstitial pneumonia and have certain evaluation value for the patient s condition and prognosis

KEY WORDS Krebs von den Lungen 6 Lactate dehydrogenase Connective tissue disease Interstitial pneumonia

1

1.2 2013 /

2 8

71 66

2

3

usual interstitial pneumonia UIP  
nonspecific interstitial pneumonia NSIP

8

9

10

6 krebs von den Lungen 6 KL 6 2019 6

lactate dehydrogenase LDH C 30

C reactive protein CRP 4 5

KL 6 LDH

1.3

KL 6

BECKMAN

COULTER DXI800 LDH

1.1

COBASC501 CRP DD

2014 1 2016 6

137 23~79

BIO RAD iMark680

51.27±13.86 22 115

1.4

45 21~75

5 mL 2 000 rpm

49.42±15.61 12

10 min KL 6

33

LDH CRP D D dimer DD

KL 6 LDH

CRP DD

1.5

SPSS 17.0

67

t

SNK q  
<sup>2</sup> P<0.05  
 2.2  
 2.3  
 2.1  
 KL 6 LDH CRP DD  
 P<0.05  
 P<0.05 DD  
 P<0.05  
 KL 6 LDH CRP  
 1 3

Table 1 Comparison of serum markers levels between 2 groups

	n	KL 6 U/mL	LDH U/L	CRP mg/L	DD mg/L
	71	1263.51±354.27 <sup>ab</sup>	362.59±124.64 <sup>ab</sup>	31.46±10.57 <sup>ab</sup>	1.65±0.47 <sup>ab</sup>
	66	249.35±76.31 <sup>a</sup>	215.46±65.87 <sup>a</sup>	3.69±1.16 <sup>a</sup>	0.72±0.23 <sup>a</sup>
	45	137.64±32.68	151.97±37.23	1.63±0.52	0.15±0.04
F		475.509	87.710	401.649	316.400
P		<0.001	<0.001	<0.001	<0.001

<sup>a</sup>P<0.05

<sup>b</sup>P<0.05

Table 2 Comparison of serum markers in IP patients with different imaging types

	n	KL 6 U/mL	LDH U/L	CRP mg/L	DD mg/L
UIP	29	1184.25±385.21	327.64±110.39	28.58±9.42	1.54±0.40
NSIP	24	1297.46±321.58	359.42±137.85	26.35±8.23	1.77±0.54
	18	1032.67±330.74	378.31±128.72	33.12±11.49	1.63±0.46
F		2.921	0.993	2.591	1.604
P		0.061	0.376	0.082	0.209

Table 3 Comparison of serum markers levels in IP patients with different imaging grades

	n	KL 6 U/mL	LDH U/L	CRP mg/L	DD mg/L
	20	1084.32±316.58	289.67±87.69	22.37±7.10	1.32±0.35
	26	1286.37±342.65	329.61±95.28	32.54±10.34 <sup>a</sup>	1.61±0.50
	25	1350.14±369.74 <sup>a</sup>	435.19±145.32 <sup>ab</sup>	39.45±12.62 <sup>ab</sup>	1.93±0.63 <sup>ab</sup>
F		3.491	10.157	14.822	7.839
P		0.036	<0.001	<0.001	<0.001

<sup>a</sup>P<0.05

<sup>b</sup>P<0.05

2.4  
 KL 6 LDH CRP DD  
 P<0.05 5  
 LDH CRP DD  
 P<0.05 4

2.5

4  
 Table 4 Comparison of serum markers levels in IP patients with different disease activity

	n	KL 6 U/mL	LDH U/L	CRP mg/L	DD mg/L
	42	1372.82±385.29	416.57±131.39	37.69±11.48	1.98±0.61
t	29	1156.31±293.47	329.64±98.57	26.34±8.23	1.39±0.35
P		2.555	3.021	4.570	4.696
		0.013	0.004	<0.001	<0.001

KL 6 LDH CRP DD

12

Wakamatsu<sup>18</sup>

KL 6 KL 6

KL 6 KL 6 KL 6 LDH CRP DD

KL 6

13 LDH CRP

14 DD

15 DD LDH KL 6

16

KL 6

3

UIP NSIP

Wang<sup>17</sup>

KL 6

KL 6 LDH CRP DD

5 2018 19 2 101.  
 12  
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1538

D D DVT  
 DVT  
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# CEACAM1

1 2 1

118 2018 04 2020 4 1 CEACAM1 55

63 CEACAM1 19 Cyfra21 1

3 Cal 3 CEA 9 MMP 9

A1 FOXA1 ObR PCNA BCL6

1 BCORL1 S100 A4 S100A4 AT 1 SATB1 Twist

1 Twist1 CEACAM1 Cyfra21 1 Cal 3 CEA MMP 9 FOXA1 ObR PCNA BCORL1

S100A4 SATB1 Twist1 CEACAM1

P<0.05 Cyfra21 1 Cal 3 CEA MMP 9 FOXA1

ObR PCNA BCORL1 S100A4 SATB1 Twist1

P<0.05 Spearman CEACAM1 Cyfra21 1 Cal 3 CEA MMP 9 FOXA1 ObR

PCNA BCORL1 S100A4 SATB1 Twist1 P<0.05

CEACAM1

1

## Expression level of CEACAM1 in fine needle aspiration tissue of thyroid cancer and its correlation with tumor malignancy

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**ABSTRACT** Objective To analyze the expression level of carcinoembryonic antigen related cell adhesion molecule 1 CEACAM1 in fine needle aspiration tissue of thyroid cancer and its correlation with tumor malignancy. Methods A total of 118 patients who underwent fine needle aspiration biopsy of thyroid nodules in the hospital from April 2018 to April 2020 were enrolled as the research objects. According to the pathologic examination results after puncture there were 55 cases of thyroid cancer thyroid cancer group and 63 cases of benign thyroid tumor thyroid benign tumor group . The expression levels of CEACAM1 serum tumor markers cytokeratin 19 fragment Cyfra21 1 galactose hemagglutinin 3 Cal 3 carcinoembryonic antigen CEA matrix metalloproteinase 9 MMP 9 proliferation genes forkhead box protein A1 FOXA1 leptin receptor ObR proliferating cell nuclear antigen PCNA and invasion genes BCL6 co

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repressor protein 1 BCORL1 S100 calcium binding protein A4 S100A4 specific AT sequence binding protein 1 SATB1 human Twist related protein 1 Twist1 were detected. The correlation between CEACAM1 and Cyfra21 1 Cal 3 CEA MMP 9 FOXA1 ObR PCNA BCORL1 S100A4 SATB1 Twist1 was analyzed. Results The expression level and positive rate of CEACAM1 in the thyroid cancer group were higher than those in the benign thyroid tumor group. The difference was statistically significant  $P<0.05$ . The levels of serum Cyfra21 1 Cal 3 CEA and MMP 9 FOXA1 ObR PCNA BCORL1 S100A4 SATB1 and Twist1 in the thyroid cancer group were higher than those in benign thyroid tumor group. The difference was statistically significant  $P<0.05$ . Spearman correlation analysis showed that expression level of CEACAM1 was positively correlated with Cyfra21 1 Cal 3 CEA MMP 9 FOXA1 ObR PCNA BCORL1 S100A4 SATB1 and Twist1  $P<0.05$ . Conclusion CEACAM1 is highly expressed in fine needle aspiration tissue of thyroid cancer and is positively correlated with the degree of tumor malignancy.

KEY WORDS Thyroid cancer Fine needle aspiration tissue Carcinoembryonic antigen adhesion molecule 1 Tumor malignancy

6.92±1.22 mm n=63 34  
 30 30-74 51.17±6.64  
 3-15 mm 7.51±1.26 mm  
 5 90 2 P>0.05

3  
 1.2  
 1.2.1 mRNA

carcinoembryonic antigen related cell adhesion molecule CEACAM

4  
 CEACAM1 RNA cDNA  
 CEACAM1 CEACAM1 actin  
 CEACAM mRNA RNA = OD260-320 ×  
 ×0.04 μg/μL OD230 260 280 320

1  
 1.1  
 2018 4 2020 4  
 118  
 3000 r/min 10 min  
 4 mL

6  
 CEACAM1

1.2.2

Cobas e 60  
 19 serum cytokeratin 19 frag  
 ment Cyfra21 1 3 galactose hem  
 agglutinin 3 Cal 3 carcinoembryonic  
 antigen CEA 9 matrix metallo  
 proteinase 9 MMP 9

1.2.3

n=55 24 21 30-75  
 50.36±6.57 4-13 mm Western

FOXA1 ObR PCNA BCORL1 S100A4 SATB1  
Twist1

1.3

SPSS 20.0

-

2

t

n

Spearman

r

P<0.05

2

2.1

CEACAM1

CEACAM

P<0.05

1

1

CEACAM1

n %

Table 1 Comparison of CEACAM1 expression between 2 groups n %

	n	mRNA	
	55	0.99±0.07	44 80.00
	63	0.20±0.04	15 23.81
t <sup>2</sup>		76.443	24.877
P		0.000	0.000

2.2

Cyfra21 1 Cal 3 CEA

MMP 9

P<0.05

2

2.3

FOXA1 ObR PCNA

P<0.05

3

2.4

BCORL1 S100A4

SATB1 Twist1

P<0.05

4

4

Table 4 Comparison of protein expression levels of tumor invasion genes between 2 groups

	n	BCORL1	S100A4	SATB1	Twist1
	55	164.37±15.39	187.68±20.01	191.60±22.17	183.21±17.65
	63	96.83±11.27	98.16±17.03	95.86±18.24	99.02±14.33
t		27.421	26.254	25.728	28.582
P		0.000	0.000	0.000	0.000

2

Table 2 Comparison of serum tumor markers between 2 groups

n	Cyfra21 1 ng/mL	Cal 3 ng/mL	CEA μg/L	MMP 9 ng/mL
55	16.77±0.36	7.46±2.33	30.29±6.04	14.49±4.03
63	5.57±0.20	4.81±0.52	17.68±4.12	8.20±2.27
t	212.320	8.785	13.387	10.613
P	0.000	0.000	0.000	0.000

3

Table 3 Comparison of protein expression levels of tumor proliferation genes between 2 groups

n	FOXA1	ObR	PCNA
55	173.21±20.14	149.75±16.28	194.71±25.08
63	98.74±15.22	99.05±12.46	100.0±18.37
t	20.300	19.125	23.594
P	0.000	0.000	0.000

2.5 CEACAM mRNA

Cyfra21 1 Cal 3

CEA MMP 9 1FOXA1 ObR PCNA BCORL1

S100A4 SATB1 Twist1

Spearman

CEACAM mRNA

Cyfra21 1 Cal 3 CEA MMP 9 FOXA1

ObR PCNA BCORL1 S100A4 SATB1 Twist11

P<0.05

5

3

CEACAM

583

67

CEACAM1

89

CEACAM1

10

11

CEACAM1

CEACAM1

5 CEACAM mRNA Cyfra21 1 Cal 3 CEA  
 MMP 9 1FOXA1 ObR PCNA BCORL1 S100A4  
 SATB1 Twist1

Table 5 Correlation analysis between CEACAM mRNA expression and Cyfra21 1 Cal 3 CEA MMP 9 1FOXA1 ObR PCNA BCORL1 S100A4 SATB1 Twist1

	CEACAM mRNA	
	r	P
Cyfra21 1	0.377	<0.001
Cal 3	0.365	<0.001
CEA	0.401	<0.001
MMP 9	0.369	<0.001
FOXA1	0.358	<0.001
ObR	0.366	<0.001
PCNA	0.381	<0.001
BCORL1	0.388	<0.001
S100A4	0.390	<0.001
SATB1	0.405	<0.001
Twist1	0.403	<0.001

CEACAM1

CEACAM Cyfra21 1 Cal 3 CEA  
 MMP 9 FOXA1 ObR PCNA BCORL1 S100A4  
 SATB1 Twist11

1

12

13

CEACAM1

CEACAM1

Giovanella

14

Cyfra21.1

Cyfra21 1

19

Cal 3

CEA

MMP9

15

Cyfra21 1 Cal 3 CEA

16

FOXA1 ObR PCNA

17

BCORL1 S100A4 SATB1 Twist1

BCORL1 1S100A4

SATB1

Twist1

NF kB

18

# BSP SOST Ca<sup>2+</sup>

Ca<sup>2+</sup> MHD 2018 6 2020 3 BSP SOST  
MHD 70 3 AACs AACs MHD / AACs 4  
AACs>4 LDL C HDL C Ca<sup>2+</sup> TG TC  
SOST 70 MHD / 28 40.00% BSP 42 60.00%  
/ Pearson AACs Ca<sup>2+</sup> BSP SOST P<0.05  
Logistic OR=1.204 95%CI 1.086-1.336 BSP OR=1.445 95%CI  
1.073-1.946 SOST OR=2.252 95%CI 1.353-3.749 MHD  
P<0.05 MHD BSP SOST  
MHD

## Relationship between serum BSP SOST Ca<sup>2+</sup> levels and abdominal aortic calcification in patients undergoing maintenance hemodialysis

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**ABSTRACT** Objective To explore the relationship between serum bone sialoprotein BSP sclerostin SOST Ca<sup>2+</sup> levels and abdominal aortic calcification in patients undergoing maintenance hemodialysis MHD . Methods Seventy patients who underwent regular MHD in blood purification center in the hospital from June 2018 to March 2020 were enrolled. The abdominal aortic calcification was evaluated by abdominal lateral X ray films. The abdominal aortic calcification scores AACs were calculated. According to different AACs MHD patients were divided into the non/mild calcification group AACs not higher than 4 points and the moderate severe calcification group AACs higher than 4 points . The dialysis time was over 3 months. Before dialysis the levels of hemoglobin albumin triglyceride TG total cholesterol TC low density lipoprotein cholesterol LDL C high density lipoprotein cholesterol HDL C glycated hemoglobin blood lipid Ca<sup>2+</sup> phosphorus BSP and SOST were measured and compared. Results Among the 70 patients with MHD 28 cases 40.00% were in the non/mild calcification group and 42 cases 60.00% were in the moderate severe calcification group. The differences in age dialysis age Ca<sup>2+</sup> phosphorus BSP and SOST

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were statistically significant between the non/mild calcification group and the moderate severe calcification group  $P<0.05$ . Pearson correlation analysis showed that AACs were significantly positively correlated with age dialysis age calcium phosphorus BSP and SOST  $P<0.05$ . Multivariate Logistic regression analysis indicated that the age of dialysis OR=1.204 95%CI 1.086-1.336 increased level of BSP OR=1.445 95% CI 1.073-1.946 and increased level of SOST OR=2.252 95% CI 1.353-3.749 were independent risk factors for moderate to severe abdominal aortic calcification in MHD patients  $P<0.05$ . Conclusion MHD patients are mostly accompanied with abdominal aortic calcification. Long dialysis age and increased levels of BSP and SOST are independent risk factors for moderate and severe calcification in MHD patients.

KEY WORDS Maintenance hemodialysis Abdominal aortic calcification Bone sialoprotein Sclerostin

MHD maintenance hemodialysis triacylglycerol TG  
 ESRD end stage renal disease total cholesterol TC low  
 ESRD 80% 1 MHD density lipoprotein LDL C high  
 MHD 50% 2 MHD Ca<sup>2+</sup> BSP SOST BSP SOST  
 MHD 3 MHD bone sialoprotein 2017  
 BSP sclerostin SOST 45 MHD X  
 MHD 1 4  
 BSP SOST Ca<sup>2+</sup> MHD MHD 6  
 MHD AACs abdominal aortic calcification score AACs  
 MHD AACs 0-24 7 MHD AACs 4  
 1 AACs>4  
 1.1 1.3  
 2018 6 2020 3 SPSS 20.0  
 70 18 MHD n % 2 Pearson  
 3 MHD P<0.05 Logistic  
 27 1 2  
 43 29-75 56.52±7.84 2.1  
 5-70 47.29±10.71 19 70 MHD  
 26 13 28 40.00% 42 60.00%  
 12 /  
 1.2 TC TG HDL C LDL C  
 MHD P>0.05 Ca<sup>2+</sup> BSP SOST  
 10 mL P<0.05 1

1

Table 1 Comparison of general information and laboratory examination results between 2 groups

	/	n=28	n=42	2/ t	P
		18	25	0.161	0.688
		10	17		
		53.21±7.96	58.73±7.62	2.917	0.005
		36.98±9.45	54.17±12.13	6.323	0.000
g/L		109.91±18.83	112.34±16.98	0.562	0.576
g/L		37.05±5.10	35.87±4.74	0.990	0.326
		11	18	0.088	0.766
%		5.28±1.04	5.51±1.13	0.861	0.392
TC mmol/L		3.79±0.84	3.85±0.80	0.301	0.764
TG mmol/L		1.56±0.49	1.62±0.44	0.534	0.595
HDL C mmol/L		1.52±0.55	1.43±0.47	0.733	0.466
LDL C mmol/L		2.27±0.60	2.46±0.71	1.165	0.248
Ca <sup>2+</sup> mmol/L		2.06±0.24	2.20±0.27	2.220	0.030
mmol/L		1.67±0.48	1.90±0.44	2.066	0.043
BSP ng/mL		16.81±4.62	23.97±3.76	7.118	0.000
SOST ng/mL		2.10±0.68	2.87±0.74	4.403	0.000

2.2 AACs

Pearson

Ca<sup>2+</sup> BSP SOST AACs P<0.05

TC TG

HDL C LDL C P>0.05 2

2 AACs

Table 2 Correlation analysis between AACs and clinical indicators

	r	P
	0.487	0.004
	0.425	0.006
g·L <sup>-1</sup>	0.127	0.301
g·L <sup>-1</sup>	-0.208	0.249
%	0.153	0.290
TC mmol·L <sup>-1</sup>	0.096	0.435
TG mmol·L <sup>-1</sup>	0.132	0.297
HDL C mmol·L <sup>-1</sup>	-0.165	0.276
LDL C mmol·L <sup>-1</sup>	0.226	0.218
Ca <sup>2+</sup> mmol·L <sup>-1</sup>	0.359	0.037
mmol·L <sup>-1</sup>	0.376	0.030
BSP ng·mL <sup>-1</sup>	0.502	0.003
SOST ng·mL <sup>-1</sup>	0.464	0.004

2.3 MHD

Logistic

MHD

1= 0= /

1 /

Logistic

BSP SOST

MHD

P<0.05 3

3 MHD Logistic

Table 3 Logistic regression analysis of multiple factors influencing abdominal aortic calcification in MHD patients

	SE	Wald/ 2	OR	95%CI	P
	0.186	0.053	12.316	1.204 1.086-1.336	0.000
BSP	0.368	0.152	5.861	1.445 1.073-1.946	0.016
SOST	0.812	0.260	9.754	2.252 1.353-3.749	0.002

3

ESRD

ESRD 11 200-300

ESRD

ESRD

ESRD

ESRD

80-90%

MHD

MHD

smooth muscle cell VSMC

VSMC

BST

AACs

BSP SOST

Logistic

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ESRD

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ESRD

ESRD

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ESRD

ESRD 11 200-300

ESRD

ESRD

ESRD

ESRD

80-90%

MHD

MHD

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VSMC

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AACs

BSP SOST

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ESRD

ESRD 11 200-300

ESRD

ESRD

ESRD

ESRD

80-90%

MHD

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VSMC

BST

AACs

BSP SOST

Logistic

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ESRD

ESRD 11 200-300

ESRD

ESRD

ESRD

ESRD

80-90%

MHD

MHD

smooth muscle cell VSMC

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BST

AACs

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	SE	Wald/ 2	OR	95%CI	P
	0.186	0.053	12.316	1.204 1.086-1.336	0.000
BSP	0.368	0.152	5.861	1.445 1.073-1.946	0.016
SOST	0.812	0.260	9.754	2.252 1.353-3.749	0.002

3

ESRD

ESRD 11 200-300

ESRD

ESRD

ESRD

ESRD

80-90%

MHD

MHD

smooth muscle cell VSMC

VSMC

BST

AACs

BSP SOST

Logistic

Table 3 Logistic regression analysis of multiple factors influencing abdominal aortic calcification in MHD patients

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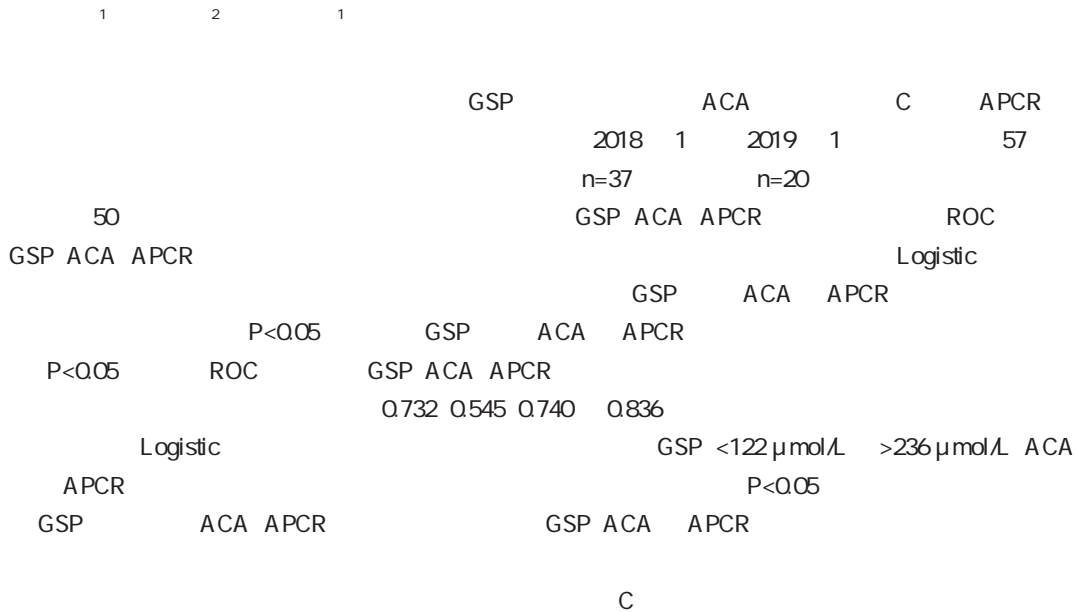
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3

ESRD



# GSP ACA APCR



## Predictive value of GSP ACA and APCR in the short term prognosis of patients with fracture of tibial plateau

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**ABSTRACT** Objective To study the predictive value of glycosylated serum proteins GSP anti cardiolipin antibody ACA and activated protein C resistance APCR in the short term prognosis of patients with fracture of tibial plateau. Methods The clinical data of 57 patients with fracture of tibial plateau fracture group admitted in the Department of Orthopedics From January 2018 to January 2019 were analyzed and they were divided into the effective group n=37 and the ineffective group n=20 according to prognosis. In addition 50 healthy people who underwent health examinations in this hospital during the same period were selected as the control group. The differences in GSP ACA and APCR indexes between different populations

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Logistic regression analysis was used to analyze the risk factors that affect the short term prognosis of patients with fracture of tibial plateau. Results The GSP level and positive rates of ACA and APCR in the fracture group were higher than those in the control group and the difference was statistically significant  $P < 0.05$ . The GSP level and positive rates of ACA and APCR in the effective group were lower than those in the ineffective group and the difference was statistically significant  $P < 0.05$ . The ROC curve was used to analyze the predictive value of GSP ACA APCR and the combined detection of the three indicators in the short term prognosis of patients with fracture of tibial plateau. The area under the curve of each indicator and three combined was 0.732 0.545 0.740 and 0.836 respectively and the combined detection has the best predictive value. According to the analysis of unconditional multivariate logistic regression model postoperative complications of deep vein thrombosis GSP  $< 122$  or  $> 236 \mu\text{mol/L}$  positive ACA and positive APCR are independent risk factors affecting the short term prognosis of patients with fracture of tibial plateau  $P < 0.05$ . Conclusion Patients with fracture of tibial plateau have higher GSP level and the expressions of ACA and APCR are mainly positive GSP ACA and APCR are the risk factors that affect the short term prognosis of patients with fracture of tibial plateau. The combined detection of the three indicators can be used as an effective means to predict the prognosis of patients with venous thrombosis.

KEY WORDS Glycosylated serum proteins Anti cardiolipin antibody Activated protein C resistance Fracture of tibial plateau

n=37

1

n=20

2

3

X

4

glycated serum proteins GSP

5

anti cardiolipin antibody ACA

C ac tivated protein c resistance APCR

6

GSP ACA

APCR

1

1.1

2018 1 2019 1 57

36

21 43.51 ± 5.47 Schatzker

6 23 17 10 7

1.3 F ROC n % t  
 GSP ACA APCR  
 122-236 μmol/L<sup>7</sup>  
 ACA Logistic P<0.05  
 OD > Cutoff OD Dahl 2  
 back APCR APC 2.1 GSP ACA APCR  
 Q 68<sup>8</sup> 2.2 P<0.05 2  
 1.4<sup>9</sup> GSP ACA APCR  
 2 GSP ACA APCR  
 1.5 SPSS 18.0 P<0.05 3

Table 2 Comparison of GSP ACA and APCR between 2 groups n %

	n	GSP μmol/L	ACA n=47		APCR n=47	
	57	456.71±13.17	48 84.21	9 15.79	45 78.95	12 21.05
	50	155.31±10.55	6 12.00	44 88.00	4 8.00	46 92.00
t <sup>2</sup>	-	129.425	55.560		54.010	
P	-	<0.001	<0.001		<0.001	

Table 3 Comparison of effective group and invalid GSP ACA and APCR n %

	n	GSP μmol/L	ACA n=37		APCR n=37	
	37	277.13±7.95	7 18.92	30 81.08	10 27.03	27 72.97
	20	498.35±10.82	18 90.00	2 10.00	15 75.00	5 25.00
t <sup>2</sup>	-	121.487	26.639		12.134	
P	-	<0.001	<0.001		<0.001	



Figure 1 Predictive value of GSP ACA APCR and combined detection in patients with fracture of tibial plateau

Table 5 Multi factor analysis of recent prognosis of patients with tibial plateau fracture

			Wald/ <sup>2</sup>	OR	95%CI	P		
			0.713	0.144	4.331	2.04	1.54-2.71	0.013
			0.846	0.713	4.651	2.33	0.58-9.43	0.611
			0.853	0.515	4.765	2.35	0.86-6.44	0.753
GSP <122 μmol/L	>236 μmol/L	122-236 μmol/L	0.964	0.135	4.953	2.62	2.01-3.42	<0.001
	ACA		0.756	0.144	4.437	2.13	1.61-2.82	<0.001
	APCR		0.748	0.245	4.356	2.11	1.31-3.42	<0.001

3 ACA ACA

ACA

APCR

C

10 15

Takeshi <sup>16</sup>

APCR

APCR

Logistic

GSP ACA

11

TNF GSP IL 6 APCR

12

GSP ACA APCR

APCR

1

CT

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2018 30 8 96 99.

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4

Johnson <sup>13</sup>

ACA

ACA

ACA

14

G 17 C

J .

2017 5 1 51 54.

1560

## TRDMT 1 CEACAM 1

tRNA 1 TRDMT 1 1  
 CEACAM 1 62  
 <5 cm TRDMT 1  
 CEACAM 1 LMVD  
 TRDMT 1 CEACAM 1 61.29% 64.52%  
 27.42% 30.65% LMVD  
 $t=22.976$   $P<0.05$   
 TRDMT 1 CEACAM 1 TNM  
 $P<0.05$  TRDMT 1  $P<$   
 0.05 TRDMT 1 CEACAM 1 LMVD TRDMT 1 CEACAM 1  
 $P<0.05$  Logistic TRDMT 1 CEACAM 1  
 $P<0.05$  TRDMT 1 CEACAM 1 LMVD  
 TRDMT 1 CEACAM 1  
 tRNA 1 1

### Expression of TRDMT 1 and CEACAM 1 in colorectal cancer and its correlation with tumor biological characteristics

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**ABSTRACT** Objective To analyze the expression of tRNA aspartic acid methyltransferase1 TRDMT 1 carcinoembryonic antigen related cell adhesion molecule 1 CEACAM 1 in colorectal cancer and its correlation with tumor biological characteristics. Method The clinical data of 62 patients with colorectal cancer were selected. The normal intestinal mucosal tissues less than 5 cm from the tumor tissue of patients in this group were selected as a reference. The differences in lymphatic micro vessel density LMVD of patients with different expressions of TRDMT 1 and CEACAM 1 were compared and risk factors affecting the prognosis of patients with colorectal cancer were analyzed. Results The positive expression rates of TRDMT 1 and CEACAM 1 in colorectal cancer were 61.29% and 64.52% respectively. The positive expression rates of TRDMT 1 and CEACAM 1 in normal tissues adjacent to cancer were 27.42% and 30.65% respectively which were significantly lower than those in colorectal cancer tissues and the LMVD value in colorectal cancer tissues was also significantly higher than that in normal cancer tissues  $t=22.976$   $P<0.05$  .

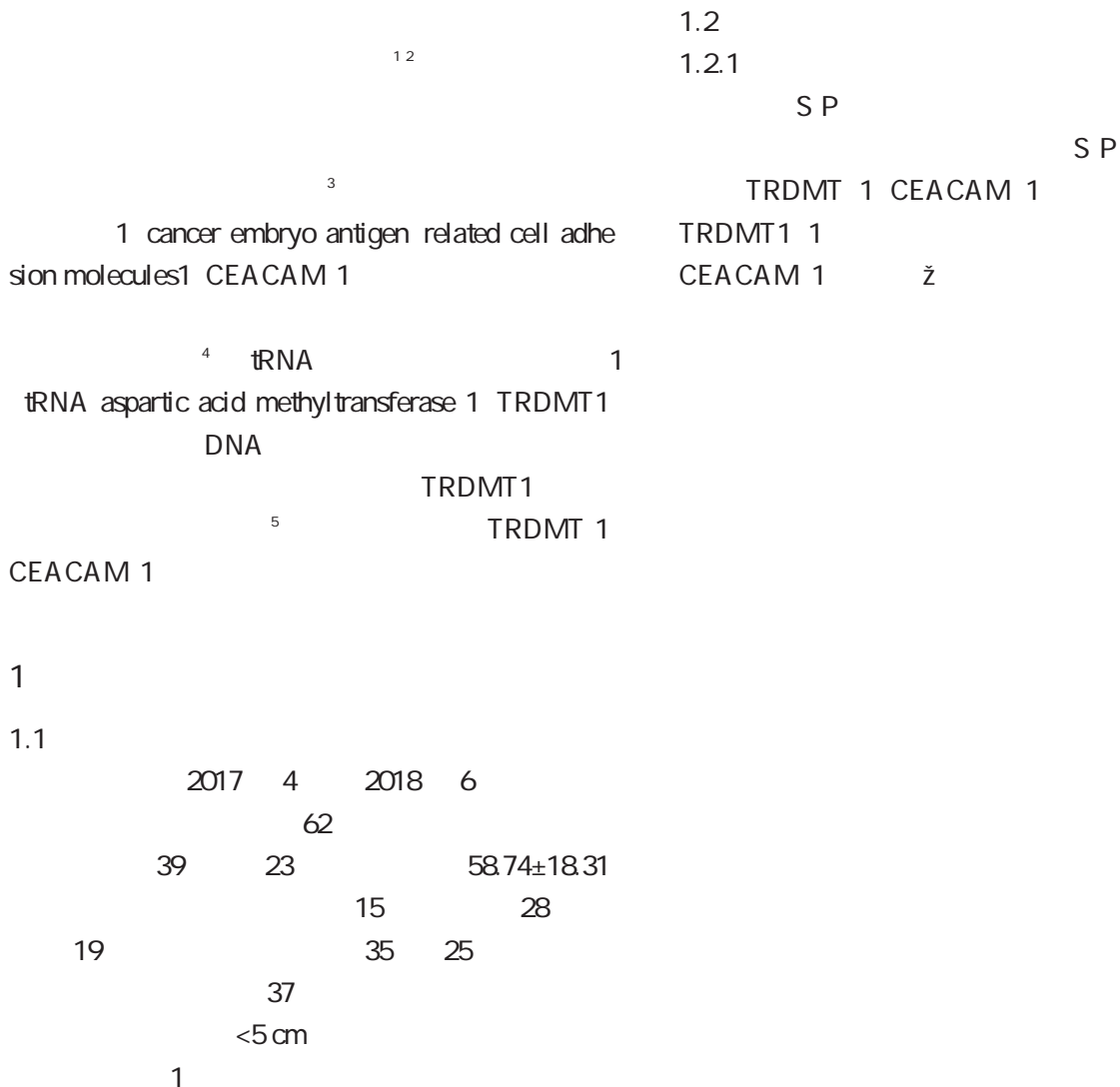
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The expressions of TRDMT 1 and CEACAM 1 in colorectal cancer tissues were related to tumor serous infiltration TNM staging liver metastasis lymph node metastasis and vascular invasion  $P<0.05$  . The expression of TRDMT 1 was closely related to the tumor diameter and the degree of histological differentiation  $P<0.05$  . The LMVD values of patients with positive expression of TRDMT 1 and CEACAM 1 in colorectal cancer were significantly higher than those with negative expression of TRDMT 1 and CEACAM 1  $P<0.05$  . Unconditional multivariate logistic regression analysis showed that the positive expressions of TRDMT 1 and CEACAM 1 were the risk factors affecting the death prognosis of patients with colorectal cancer  $P<0.05$  . Conclusion The high expression of TRDMT 1 and CEACAM 1 and the increase of LMVD are closely related to the biological behavior and progression of colorectal cancer. Clinically patients can be evaluated and monitored for prognosis based on the expression of TRDMT 1 and CEACAM 1.

KEY WORDS tRNA aspartic acid methyltransferase 1 Cancer embryo antigen related cell adhesion molecules1 Colorectal cancer Tumor biological characteristics



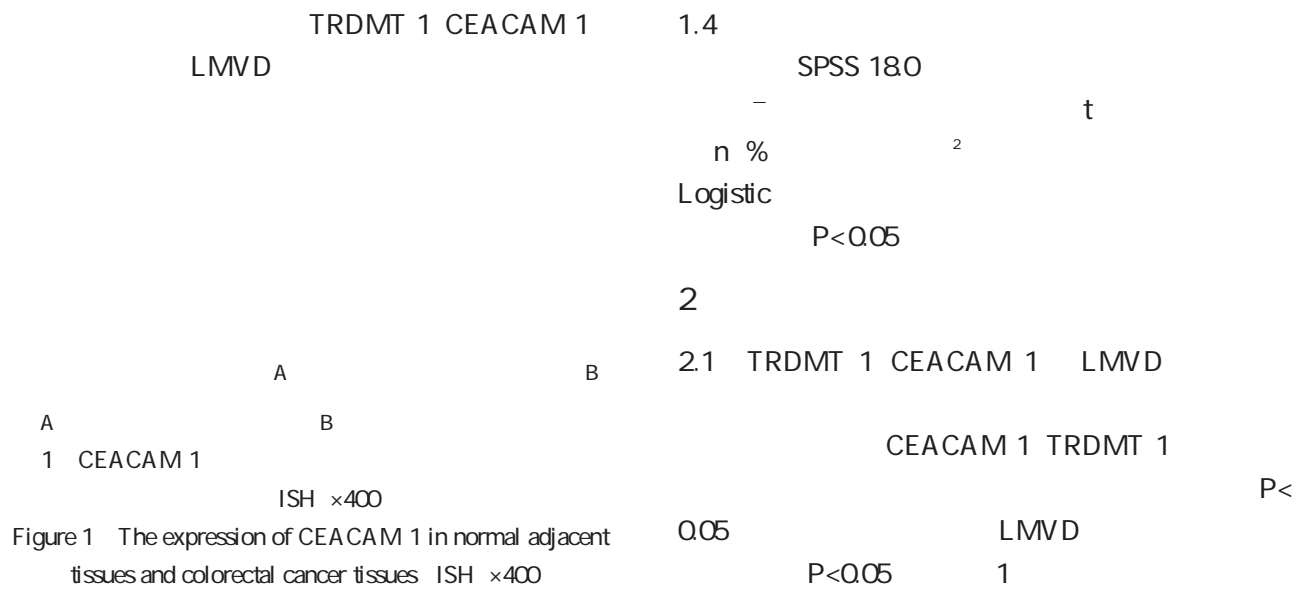


Table 1 Expressions of TRDMT1 CEACAM 1 and LMVD in different tissues

	n	CEACAM 1		TRDMT 1		LMVD
		n	%	n	%	
t <sup>2</sup>	62	22	35.48	40	64.52	14.29±2.79
P	62	43	69.35	19	30.65	4.42±1.14
			14.259		14.409	22.976
			0.001		0.001	0.001

2.2 TRDMT 1 CEACAM 1 P<0.05 4

TRDMT 1 CEACAM 1 3

TNM

P<0.05 CEACAM 1 5 60

P>0.05 TRDMT 1 8 CEA CA 75

P<0.05 TRDMT 1

CEACAM 1

TNM

P<0.05 2 9

2.3 TRDMT 1 CEACAM 1 DNA

LMVD TRDMT 1 DNA

TRDMT 1 CEACAM 1 LMVD 5

TRDMT 1 CEACAM 1 DNA<sup>10</sup> TRDMT 1 DNA

P<0.05 3 DNA

2.4

62

13 20.97% 49 79.03% 11

TNM LMVD TRDMT 1

TRDMT 1 CEACAM 1

2 TRDMT 1 CEACAM 1

n %

Table 2 Correlation between the expression of TRMT1 and CEACAM 1 in colorectal cancer and its clinicopathological factors

		n %								
		n	TRDMT 1	n=38	<sup>2</sup>	P	CEACAM 1	n=40	<sup>2</sup>	P
TNM	5 cm	35	30 78.95		13.252	0.001	23 57.50		0.050	0.822
	<5 cm	27	8 21.05				17 42.50			
		43	30 78.95		4.250	0.039	32 80.00		6.010	0.014
		19	8 21.05				8 20.00			
	+	17	7 18.42		3.994	0.046	7 17.50		5.573	0.018
	+	45	31 81.58				33 82.50			
		15	10 26.32				9 22.50			
		28	12 31.58		8.400	0.015	18 45.00		0.261	0.878
		19	16 42.11				13 32.50			
		18	15 39.47		5.194	0.023	16 40.00		6.581	0.010
		44	23 60.53				24 60.00			
		35	27 71.05		8.513	0.004	28 70.00		8.417	0.004
		27	11 28.95				12 30.00			
		22	18 47.37		6.057	0.014	18 45.00		4.459	0.035
	40	20 52.63				22 55.00				

3 TRDMT 1 CEACAM 1

TRDMT 1

LMVD -

Table 3 Relationship between the expression of TRMT 1 and CEACAM 1 and LMVD in colorectal cancer -

	LMVD	t	P
CEACAM 1	9.91±2.14 17.36±2.06	13.440	0.001
TRDMT 1	9.81±2.79 16.80±2.47	10.322	0.001

CEACAM 1

Okegawa T <sup>12</sup>

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Table 4 Analysis of risk factors affecting prognosis and death of colorectal cancer patients

			OR	95%CI	P	OR	95%CI	P
TNM	5 cm	<5 cm	1.103	0.867-1.404	0.835			
			1.709	1.093-2.672	0.004	1.848	1.038-3.288	0.014
	+		1.881	1.003-3.530	0.012	1.480	1.138-1.924	0.023
	+	+	1.793	1.034-3.110	0.009	1.560	1.052-2.314	0.006
TRDMT 1			1.619	1.024-2.562	0.026	1.589	1.047-2.412	0.007
CEACAM 1			1.690	1.105-2.587	0.015	1.828	1.050-3.183	0.019
LMVD			1.744	1.039-2.925	0.008	1.802	1.074-3.024	0.025
			1.728	1.030-2.899	0.016	1.831	1.043-3.214	0.003

CEACAM 1 L CEACAM 1 S

<sup>13</sup>

TRDMT 1 CEACAM 1

TRDMT 1

LMVD

CEACAM 1

TRDMT 1

<sup>14</sup>

Arabzadeh A <sup>15</sup>

CEACAM 1

TRDMT 1

CEACAM 1

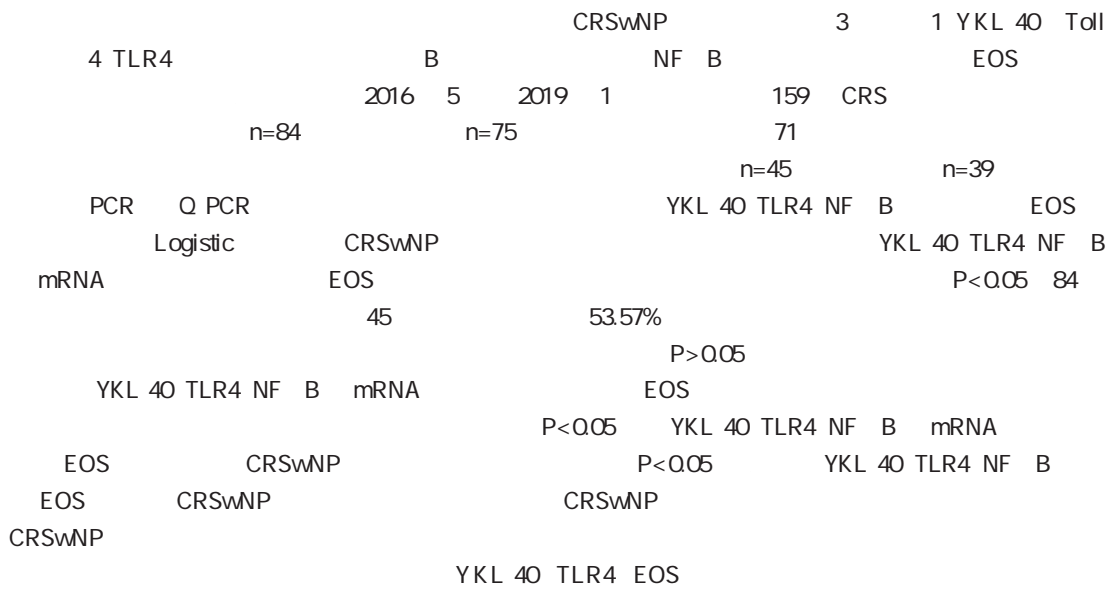
LMVD

	TRD	7			
MT 1 CEACAM 1			J .	2019 16	
				2 60 62	
TRDMT 1 CEACAM 1		8	Zaidi S Chen J Korkut A et al. Mo1971 Crosstalk Between Ceacam and Tgf Signaling Pathways and their Role in Colorectal Cancer J . Gastroenterology 2018 154 6 S 868		
LMVD		9		. CEACAM1	
			VEGF MVD J .	2017 17	
			6 553 555.		
		10		. CEACAM 1	
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2	. ctDNA		J .	2018 23 6	
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				J .	
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	Meta J .	2015 40 3			
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		2017 27 6 1337 1340			
10					
	Meta J .	2015 21 11 993 997.			
11					

# CRSwNP YKL 40 TLR4



Correlation between *YKL-40* *LR4* gene expression eosinophi level and relapse  
in patients with chronic x o m R/

relapse of patients with CRSwNP. Results The YKL 40 TLR4 mRNA level of NF B and the percentage of EOS in peripheral blood and tissues in the polyps group were higher than those in the non polyps group and the control group. The difference was statistically significant  $P<0.05$  . As of the follow up date 84 patients in the polyp group had 45 recurrences and the recurrence rate was 53.57% There was no significant difference in age gender ratio of lymphocyte in peripheral blood running nose score and nasal congestion score between the relapse group and the non relapse group. However the proportion of asthma dysosmia score YKL 40 TLR4 mRNA level of NF B and the percentage of EOS in peripheral blood and tissues in the relapse group were higher than those in the non relapse group and the proportion of lymphocytes in tissue and plasma cells in tissue were lower than those in the relapse group and the difference was statistically significant  $P<0.05$  . High expression levels of YKL 40 TLR4 and mRNA of NF B and increased EOS in tissue are independent risk factors that affect the relapse of CRSwNP patients  $P<0.05$  . Conclusion The YKL 40 TLR4 NF B and EOS rate in tissue are highly expressed in patients with CRSwNP which can used as independent risk factors affecting the prognosis relapse of CRSwNP and is of great significance for clinical prediction of the disease progression of CRSwNP patients.

KEY WORDS Chonic sinusitis with nasal polyp YKL 40 TLR4 EOS

chonic sinusitis CRS 159 CRS  
12 n=84 n=75 £ œ` "t i•

<sup>1</sup> CRS  
chonic sinusitis with nasal polyp CRSwNP CRS

CRSwNP  
eosinophi  
EOS EOS EOS  
<sup>2</sup> 3 1  
chitinase 3 like protein 1 YKL 40  
<sup>3</sup> Toll 4  
toll like receptor 4 TLR4  
B  
nuclear factor kappa B NF B  
<sup>4</sup> CRS  
YKL 40 TLR4 EOS  
CRSwNP

1

1.1

2016 5 2019 1

3 YKL 40 TLR4 NF B 1.2.3  
- 70

RNA PCR TRIZOL 2020 4  
RNA Invitrogen 5  
RNA cDNA cDNA 0 0-3 4-7 8-10 VAS 6  
PCR 1.3  
SPSS 18.0  
YKL 40 5 - t F  
TGAG GCATCGCAATGTAAG 3 5 n % 2  
AAGGGGAAG TAGGATAGGGG 3 TLR4 CRSwNP Logistic  
5 TGTC CTCCACTCCAGGTAAGT 3 P<0.05  
5 GATTGCT CAGACCTGGCAGTT 3 NF 2  
B 5 TCCAGAAGTATTTCAACCA  
CAG 3 5 GCCT TCACATACATAACGGA 2.1 3 YKL 40 TLR4 NF B  
3 PCR 38 EOS  
90 60 s 92 30 s 56 30 s 74 30 s YKL 40 TLR4 NF B mRNA  
3 Ct EOS P<0.05 2  
EOS 2 3 YKL 40 TLR4 NF B mRNA EOS -

Table 2 Comparison of YKL 40 TLR4 NF B mRNA levels and tissue EOS proportion in the 3 groups

	n	YKL 40mRNA	TLR4mRNA	NF B mRNA	EOS %	EOS L <sup>1</sup> ×10 <sup>6</sup>
	84	0.97±0.31	0.93±0.15	0.89±0.13	4.97±1.57	0.31±0.12
	75	0.71±0.25	0.67±0.13	0.65±0.11	3.21±1.56	0.14±0.05
	71	0.49±0.13	0.43±0.12	0.31±0.05	2.19±1.03	0.07±0.01
F		73.67	265.94	595.57	76.50	196.26
P		<0.001	<0.001	<0.001	<0.001	<0.001

2.2 CRSwNP EOS CRSwNP  
84 P<0.05 4  
53.57% n=45 3  
n=39

2.3 CRSwNP CRSwNP  
P>0.05 CRSwNP  
YKL 40 TLR4 NF B mRNA YKL 40 18  
EOS

2.4 P<0.05 3 CRSwNP 7  
YKL 40 TLR4 NF B mRNA 8 YKL 40

3 CRSwNP

Table 3 Single factor analysis of the influence on the recurrence of CRSwNP patients

	n=45	n=39	t/ <sup>2</sup>	P
	23	15		
	22	14	0.003	0.959
	47.33±5.11	47.51±5.27	0.159	0.874
%	35.86±5.31	34.89±5.36	0.831	0.408
	7.45±1.05	7.37±1.13	0.336	0.738
	5.89±2.13	5.21±2.01	1.498	0.138
n	13	3	6.088	0.014
	4.95±1.03	1.24±0.86	17.757	0.001
YKL 40mRNA	1.13±0.51	0.85±0.43	2.697	0.009
TLR4mRNA	1.19±0.23	0.91±0.15	6.496	0.001
NF B mRNA	1.31±0.65	0.89±0.51	3.258	0.002
EOS %	5.23±1.57	4.55±1.39	2.087	0.040
EOS L <sup>-1</sup> ×10 <sup>9</sup>	0.55±0.13	0.29±0.11	9.810	0.001

4 CRSwNP

Table 4 Multivariate analysis of the influence of recurrence in crswnp patients

			WALD/ <sup>2</sup>	OR	95%CI	P
	0.931	0.615	1.339	2.537	0.760-8.469	1.307
	1.137	0.953	2.546	3.117	0.484-20.184	1.751
YKL 40MRNA	1.697	0.513	4.156	5.458	1.997-14.917	0.001
TLR4MRNA	1.791	0.413	4.798	5.995	2.669-13.470	0.001
NF BMRNA	1.635	0.535	4.681	5.129	1.798-14.638	0.001
EOS	1.371	0.941	2.591	3.939	0.623-24.912	1.395
EOS	1.659	0.219	5.311	5.254	3.420-8.071	0.001

## CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK

100 SLE CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK  
 2017 3 2019 6 145 SLE  
 SLE SLEDAI CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK  
 CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK SLE  
 P<0.05 C3 C4 CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK  
 P<0.05 SLEDAI C CRP ESR dsDNA C1q  
 SLEDAI CRP P<0.05 C3 C4 CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK  
 C3 ESR dsDNA CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK SLE Logistic P<  
 0.05 ROC CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK SLE  
 0.752 0.794 144.08  $\mu$ L 11.44% SLE  
 CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK SLE  
 SLE

### Clinical significance of changes in peripheral blood CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK cells in patients with systemic lupus erythematosus

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**ABSTRACT** Objective To explore the changes of peripheral blood CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK cells in patients with systemic lupus erythematosus (SLE) and their clinical significance. Methods A total of 145 patients with SLE who were admitted to the Department of Rheumatology and Nephrology in the hospital between March 2017 and June 2019 and 100 healthy subjects were selected as the research group. The levels of peripheral blood CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK cells in both groups were measured and clinical indexes of patients with SLE were collected. Patients were divided into active group and inactive group according to the SLEDAI score. The levels of peripheral blood CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK cells in different groups were compared and their correlation with clinical parameters in patients with SLE was analyzed. Results CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK cell count and percentage in the control group were high than those in the SLE group. The differences of CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK

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cell count and percentage between the groups were statistically significant  $P < 0.05$ . Complement C3 and C4 levels CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup> NK cell count and percentage in the active group were lower than those in the inactive group  $P < 0.05$ . SLEDAI score C reactive protein CRP erythrocyte sedimentation rate ESR anti dsDNA antibody and anti C1q antibody levels in the active group were higher than those in the inactive group  $P < 0.05$ . *Pearson* correlation analysis showed that levels of CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup> NK cells were negatively correlated with SLEDAI score and CRP levels  $P < 0.05$  but were positively correlated with complement C3 and C4 levels  $P < 0.05$ . Multivariate *Logistic* analysis showed that complement C3 ESR anti dsDNA antibody CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup> NK cell count and percentage were independently correlated with the activity of SLE  $P < 0.05$ . ROC curve showed that the area under the curve values of CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup> NK cell count and percentage for evaluating the activity of SLE were 0.752 and 0.794 respectively. When the cut off values were 144.08 cells/ $\mu$ L and 11.44% the Youden index was the largest. Conclusion There is a decrease in CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup> NK cells in SLE patients and the number is closely related to the activity of SLE patients. Therefore the number of CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup> NK cells may provide a certain reference for the evaluation of SLE patients.

KEY WORDS Systemic lupus erythematosus Natural killer cells Systemic lupus erythematosus disease activity index

systemic lupus erythemato

sus SLE

SLE

100

SLE

SLE

10 135 25-

natural killer cell NK

44 35.12 $\pm$ 9.45 SLE

SLE disease activity index SLEDAI 6

SLEDAI 5 57

NK

SLEDAI <5 88 7

93 22-46 35.01 $\pm$ 9.35 SLE

SLE NK

P > 0.05

SLE

SLE

3 4 1.2

CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK NK

1.2.1

CD3<sup>+</sup>CD16<sup>+</sup>CD56<sup>+</sup>NK

SLE

SLE

C3 C4 C C reactive pro

tein CRP erythrocyte sedimentation rate

ESR c1q dsDNA

1

1.2.2 NK

1.1

SLE

2017 3 2019 6

SLE

145

EDTA

SLE

1 mL BD Trucount

20 $\mu$ L 6

50 $\mu$ L

15 min 450 $\mu$ L BD

5

FACS 15 min P<0.05  
 mindray BriCyte E6  
 MRFlow 6 2  
 CD3 FITC CD16 PE 2.1 SLE CD3 CD16<sup>+</sup>CD56<sup>+</sup>  
 CD56 PE CD4 PE Cy7 NK  
 CD8 APC Cy7 CD45 Per CP Cy5.5 CD3 CD16<sup>+</sup>CD56<sup>+</sup>NK  
 CD19 APC BD 401 228 645 μL 19.24  
 1.3 9.88 22.85 % SLE 152 66 202 /  
 SPSS 18.0 μL 11.24 4.71 15.96 %  
 n % 2 U=22.456 19.653 P<0.05  
 - t 2.2 CD3  
 ±  
 M P25%-P75% Mann Whitey C3 C4 CD3 CD16<sup>+</sup>  
 Pearson CD56<sup>+</sup>NK  
 Logistic SLE P<0.05 ESR CRP dsDNA  
 ROC SLE C1q P<0.05 1  
 1 CD3 CD16<sup>+</sup>CD56<sup>+</sup>NK

Table 1 Comparison of activity index and LEVELS of CD3 CD16<sup>+</sup>CD56<sup>+</sup>NK cells in enrolled subjects

	n=57	n=88	U/t	P
SLEDAI	7 5 8	2 1 3	10.247	<0.001
C3 g/L	0.77±0.23	1.02±0.41	4.193	<0.001
C4 g/L	0.11±0.05	0.19±0.09	6.122	<0.001
ESR mm/h	35.29 20.14 55.29	22.56 10.27 39.65	6.523	<0.001
CRP mg/L	12.01 2.14 20.36	3.26 1.11 7.96	8.956	<0.001
dsDNA IU/mL	145.25 44.18 356.93	4.55 0 66.96	6.341	<0.001
C1q RU/mL	30.24 9.86 60.23	3.88 0.41 8.56	7.456	<0.001
CD3 CD16 <sup>+</sup> CD56 <sup>+</sup> NK μL	127 55 189	188 101 296	3.748	<0.001
CD3 CD16 <sup>+</sup> CD56 <sup>+</sup> NK %	8.56 3.52 10.24	13.33 5.01 16.52	4.012	<0.001

2.3 CD3 CD16<sup>+</sup>CD56<sup>+</sup>NK 2.4 SLE  
 Pearson SLEDAI CRP P< CD3 CD16<sup>+</sup>CD56<sup>+</sup>NK Logistic C3 ESR  
 0.05 C3 C4 P<0.05 dsDNA CD3 CD16<sup>+</sup>CD56<sup>+</sup>NK  
 2 3 SLE P<0.05  
 2 CD3 CD16<sup>+</sup>CD56<sup>+</sup>NK

Table 2 Simple correlation between the level of CD3 CD16<sup>+</sup>CD56<sup>+</sup>NK cells and activity index

	CD3 CD16 <sup>+</sup> CD56 <sup>+</sup> NK	μL	CD3 CD16 <sup>+</sup> CD56 <sup>+</sup> NK	%
	r	P	r	P
SLEDAI	-0.355	<0.001	-0.363	0.001
C3 g/L	0.352	0.120	0.301	0.048
C4 g/L	0.296	0.039	0.345	0.028
ESR mm/h	-0.256	0.078	-0.196	0.204
CRP mg/L	-0.311	0.004	-0.296	0.036
dsDNA IU/mL	-0.162	0.201	-0.201	0.096
C1q RU/mL	-0.155	0.256	-0.214	0.087

Table 3 Correlation analysis between SLE patients activity and clinical indicators

			OR	95%CI	P	OR	95%CI	P
	U/L	N	0.722	0.580-0.899	0.004	0.694	0.694-0.864	<0.001
	g/L		0.774	0.513-1.168	0.224			
@ =	ESR mm/h	C	1.394	1.141-1.702	0.001	1.293	1.044-1.607	0.019
	CRP mg/L		1.239	0.975-1.573	0.080			
	dsDNA IU/mL		1.493	1.109-2.012	0.009	1.366	1.018-1.833	0.038
	C1q RU/mL		1.292	0.959-1.740	0.093			
CD3CD16 <sup>+</sup> CD56 <sup>+</sup> NK		μL	0.798	0.653-0.974	0.027	0.752	0.606-0.933	0.010
CD3CD16 <sup>+</sup> CD56 <sup>+</sup> NK		%	0.739	0.580-0.943	0.015	0.752	0.594-0.951	0.018

2.5 CD3CD16<sup>+</sup>CD56<sup>+</sup>NK SLE

ROC CD3CD16<sup>+</sup>CD56<sup>+</sup>NK SLE  
 CD3CD16<sup>+</sup>CD56<sup>+</sup>NK SLE  
 0.752 95% CI 0.674-0.829  
 0.794 95% CI 0.727-0.865 144.08  
 μL 68.2% 75.4% 11.44%  
 62.6% 76.5%  
 0.436 0.391 1

3

NK

NK

7

NK

SLE

2019 42 9 762 767.

SLEDAI 8 NK

TIM 3 J . 2020 36 3 55 58

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1564

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2018 14 4 252 255.

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2019 26 3 473 476.

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J . 2018 22 1 24 28

G4 18 J .

2020 105 33 591 2595.

2018 J . 12 CT

2019 54 2 81 100 J . 2019 33 9 918 920

6 J .

2018 15 5 69 71.

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2018 14 4 252 255.

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2019 24 10 10.

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2019 26 3 473 476.

10 J . 2019 24 4 386 389.

J . 2018 22 1 24 28

G4 18 J .

2020 105 33 591 2595.

• •

# CIP2A VCAM 1 TRF1

1 2 2 2 2 2 2 2

2A

CIP2A

1

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1.

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2

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logical grade high grade CIP2A positive and VCAM 1 positive TRF1 negative are independent risk factors that affect the death prognosis of patients with glioma P<0.05 . Conclusion The expression of CIP2A VCAM 1 and TRF1 in gliomas is related to its pathological grade and can be used as a reference index for judging the malignant degree of gliomas and evaluating its prognosis.

KEY WORDS Cancer inhibitor of protein phosphatase 2A Vascular cell adhesion molecule 1 Telomere binding protein 1 Glioma

40%~50%<sup>1</sup>  
 30 >18  
 1.2%<sup>2</sup>  
 5 5%<sup>3</sup>  
 2A cancerous 1.2  
 inhibitor of protein phosphatase 2A CIP2A  
 S P CIP2A VCAM 1  
 1 vascular cell adhesion molecule 1 VCAM TRF1 CIP2A VCAM 1  
 VCAM 1 TRF1  
 4 1 telomere re VCAM 1  
 VCAM 1 TRF1 TRF1  
 VCAM 1 TRF1  
 5 CIP2A VCAM 1 TRF1  
 7 0 1  
 2 3 2 3 2  
 1 3  
 1.1 CIP2A VCAM 1  
 TRF1 CIP2A VCAM 1  
 2016 3 2018 4 TRF1  
 77 TRF1  
 34 43 36.14± 2  
 7.08 World Health Organiza  
 tion WHO 2016  
 6 ~ 37 1.4  
 21 16 ~ SPSS 22.0  
 40 28 12 - t n %  
 23 24 18 12 2  
 5 cm 27 >5 cm 50 P<0.05  
 50 23 27 2  
 36.25±7.04 2.1 CIP2A VCAM 1 TRF1  
 P>0.05 CIP2A VCAM 1

TRF1 P<0.05 1

1 CIP2A VCAM 1 TRF1 n %

Table 1 Comparison of CIP2A VCAM 1 and TRF1 expressions between 2 groups n %

n	CIP2A	VCAM 1	TRF1
50	23 48.00	22 44.00	45 90.00
77	69 89.61	68 88.31	39 50.65
<sup>2</sup>	- 28.879	28.831	20.961
P	- 0.001	0.001	0.001

2.2 CIP2A VCAM 1 TRF1

CIP2A VCAM 1 TRF1

2 CIP2A VCAM

} 25.64

	CIP2A	<sup>2</sup>	P	VCAM 1	<sup>2</sup>	P	TRF1	? à P %7&F&Q\$ "SÜ\$	
45	32 46.38	0.038	0.846	31 45.59	0.317	0.573	18 46.15	0.011 0.915	
>45	37 53.62			37 54.41			21 53.85		
	31 44.93	0.160	0.689	28 41.18	2.094	0.148	16 41.03	0.314 0.575	
	38 55.07			40 58.82			23 58.97		
cm	5	24 34.78	0.023	0.879	25 36.76	0.738	0.390	17 43.59	2.522 0.112
	>5	45 65.22			43 63.24			22 56.41	
		30 43.48	5.565	0.018	28 41.18	11.017	0.001	29 74.36	21.910 0.001
		39 56.52			40 58.82			10 25.64	
		21 30.43	1.593	0.661	18 26.47	5.587	0.134	11 28.21	0.753 0.861
		20 28.99			23 33.82			13 33.33	
		17 24.64			15 22.06			8 20.51	
		11 15.94			12 17.65			7 17.95	

P<0.05 TRF1 P>0.05 2

2.3 19.48% 62 80.52% CIP2A VCAM 1 TRF1

P<0.05 3

2.4 logistic CIP2A VCAM 1 TRF1 >45

P<0.05 4



# fFN MMP 9 IL 6

1 2 3

10 2019 6 fFN MMP 9 IL 6 2016 86

MMP IL 6 40 ELISA fFN

P<0.05 ROC fFN MMP 9 IL 6

AUC 0.734 0.701 0.791 0.912 P<0.05

Logistic fFN 89.62 ng/mL IL 6 32.41 pg/mL MMP 9 6.96 pg/mL

P<0.05 fFN MMP 9 IL 6

fFN MMP 9 IL 6

## Correlation analysis between serum fFN MMP 9 IL 6 levels and spontaneous preterm delivery

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**ABSTRACT** Objective To analyze the correlation between serum fFN MMP 9 IL 6 levels and spontaneous preterm labor. Methods The women who underwent production inspection and labor in the hospital from October 2016 to June 2019 were enrolled as the research subjects. The 86 women undergoing spontaneous preterm labor were included in the preterm labor group while another 40 women who underwent normal labor during the same period were enrolled as the control group. The levels of serum fFN MMP and IL 6 in both groups were detected using enzyme linked immunosorbent assay ELISA . The differences in the above indexes between the two groups were analyzed. The effect of the three alone and their combination for predicting spontaneous preterm labor and independent risk factors of spontaneous preterm labor were explored. Results The contents of fFN MMP 9 and IL 6 in the preterm labor group were higher than those in the control group P<0.05 . The results of ROC curve analysis showed that AUC values of fFN MMP 9 and IL 6 for

- 1. 2017 20180695 075000
- 2. 075000
- 3. 075000

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the diagnosis of spontaneous preterm labor were 0.734 0.701 and 0.791 lower than that of their combination  
 0.912 P<0.05 <0.05

sk U p Sh g @ \$ j U ! O s s g D p 9 M B C P P J X F S F ` 1 T M Γ a c e & 3 // Ô g e > î Ô . p \* è \* ò x U G 7 i @ ò j 0 a M \$ € D % & & ! • ò p > 6 0 # ! f p ` 6 0 # ! U F 0 7 p 0 0 0 4 a

UniCel DxI800 2  
 fFN  
 MMP 9 2.1 fFN MMP 9 IL 6  
 IL 6 fFN MMP 9 IL 6  
 ThermoFisher P<0.05 2

1.3  
 SPSS 20.0  
 - t n %  
 2 ROC fFN MMP 9  
 IL 6  
 Logistic P<0.05

2.2 fFN MMP 9 IL 6

2.3

logistic

fFN MMP 9 IL 6  
ROC

ROC

fFN MMP 9 IL 6

AUC 0.912

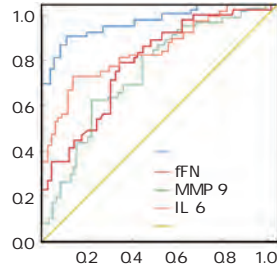
Logistic

fFN MMP 9 IL 6

fFN 89.62 ng/mL IL 6 32.41 pg/mL MMP 9 6.96 pg/mL

P<0.05 1 3

P<0.05 4



3

5%~15%

1 fFN MMP 9 IL 6

A) Q-

ROC

O E B M P O F

U'

7

Figure 1 ROC curves of fFN MMP 9 and IL 6 levels alone and combined to predict spontaneous preterm labor in patients

fFN

fFN

fFN

fFN

fFN ng/mL  
91 94

fFN

8

23

fFN

IL 6

IL 6

ROC

logistic fFN 89.62 ng/mL IL 6  
32.41 pg/mL MMP 9 6.96 pg/mL

fFN MMP 9 IL 6

fFN MMP 9 IL 6

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1573

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Periostin

2018 4 2019 10 92 DCM

Periostin T

91.30% 46 76.09%

P<0.05 P>0.05 LVEDD

LVEDD LVEF P<0.05 Periostin

Periostin BNP Hs cTnT P>0.05 Periostin

BNP Hs cTnT P<0.05

DCM Periostin

Periostin

### Analysis of the clinical value of metoprolol in the treatment of chronic heart failure caused by dilated cardiomyopathy

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**ABSTRACT** Objective To analyze the efficacy of metoprolol in the treatment of dilated cardiomyopathy of chronic heart failure and its effect on plasma periostin protein level and cardiac function. Methods The clinical data of 92DCM patients with chronic heart failure admitted to our hospital from April 2018 to October 2019 were selected. The patients were divided into the control group and the observation group according to different treatments and the control group was treated with conventional treatment. The observation group was supplemented with metoprolol. The clinical effects and the changes of cardiac function before and after treatment between the two groups were compared. The level of plasma periostin protein brain natriuretic peptide and high sensitivity troponin T were analyzed before and after treatment. Results Clinical efficacy After treatment the total effective rate of the observation group was 91.30% the total effective rate of the control group in the 46 patients was 76.09% and the total effective rate of the observation group was significantly better than that of the control group and the difference was statistically significant  $P<0.05$ . Cardiac function Before treatment the indexes of cardiac function were compared  $P>0.05$ . After treatment the levels of LVEDD LVEDS and LVEF significantly improved in 2 groups while the improvement in the observation

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group was significantly higher than that in the control group  $P < 0.05$ . Before treatment there were no significant differences in levels of plasma periostin protein BNP and Hs cTnT between the two groups  $P > 0.05$ . After treatment the levels of plasma periostin protein BNP and Hs cTnT significantly reduced in the two groups and all of them in the observation group were significantly lower than those in the control group  $P < 0.05$ . Conclusion Metoprolol therapy has a significant clinical treatment effect on patients with DCM and chronic heart failure which can effectively improve the heart function of patients and reduce the level of plasma periostin protein in patients which is worthy of wide application.

KEY WORDS Dilated cardiomyopathy of chronic heart failure Plasma periostin protein Metoprolol Cardiac function

Dilated cardiomyopathy DCM

1.2

1

23

30-50 H32025391 25 mg/ 2  
7 mg

4 2A 100 mg/d

1 Left ventricular ejection fraction LVEF Left ventricular end diastolic diameter LVEDD Left ventricular end systolic diameter

5 Periostin DCM LVEDD 3 1

6 DCM

DCM EDTA 5 mL

- 20

Periostin Brain Natriuretic Peptide BNP High sensitivity tropo ninT Hs cTnT

1 1.3

1.1

2018 4 2019 10

92 DCM Periostin 8

46

26 20 36-70 2

56.34±7.09 25

21 34-70 55.06± 1

6.37 1.4

7 SPSS 18.0

-

n % 2 t  $P < 0.05$

2 Hs cTnT P>0.05  
 2.1 Periostin BNP Hs cTnT  
 P<0.05 4  
 P>0.05 1

Table 1 Comparison of clinical data of patients in 2 groups  
 n %

n=46	26/20	56.34±7.09	7.32±1.89	20	43.48	16	34.78
n=46	25/21	55.06±6.37	7.46±1.99	16	34.78	18	39.13
t <sup>2</sup>	0.044	0.644	0.245	0.730	0.187		
P	0.834	0.523	0.808	0.393	0.666		

2.2 P<0.05 2  
 2.5 1 1  
 8.70% 2/23  
 3

Table 2 Comparison of clinical treatment effect of 2 groups of patients

n=46	20	43.48	15	32.61	11	23.91	35	76.09
n=46	24	52.17	18	39.13	4	8.70	42	91.30
<sup>2</sup>		0.348	0.425	3.903	3.903			
P		0.555	0.514	0.048	0.048			

2.3 P>0.05  
 P<0.05 3  
 9  
 0.9%<sup>10</sup>  
 DCM  
 11  
 DCM  
 1213

n=46 @  
 " 0`  
 n=46  
 " 3 0` P  
 !P y  
 )OB L 2.4 Periostin BNP 15  
 R Hs cTnT Periostin BNP  
 !



# PCI

# miR 146a Galectin 3

1 2 3 4 1

Percutaneous coronary intervention PCI

miR 146a 3 Galectin 3 507

PCI n=76 n=431

miR 146a Galectin 3 Clinical pulmonary infection score CPIS

APACHE 1 3 7 d miR 146a Galectin 3

P<0.05 1 3 7 d CPIS APACHE

P<0.05 3 d miR 146a Galectin 3 PCI 3 d CPIS APACHE P<0.05

1 3 7 d miR 146a Galectin 3 P<0.05 PCI

miR 146a Galectin 3 CPIS APACHE

miR 146a 3

## Expression of serum miR 146a and Galectin 3 in patients with pulmonary infection after PCI and their relationship with anti infection efficacy

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ABSTRACT Objective To explore the relationship between serum miR 146a and Galectin 3 expression in patients with pulmonary infection after percutaneous coronary intervention PCI and their anti infective efficacy. Methods A total of 507 patients who underwent PCI in our hospital were selected as the research subjects. They were divided into an infected group n=76 and an uninfected group n=431 according to the presence or absence of pulmonary infection. The serum miR 146a Galectin 3 clinical pulmonary infection score CPIS acute physiology and chronic health APACHE score were compared between the two groups at different times and the diagnostic value of each index was analyzed. Results The levels of serum miR 146a and Galectin 3 in the infected group were higher than those in the uninfected group at 1 day 3 days and 7 days after operation and the difference was statistically significant P<0.05 the CPIS and

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APACHE scores in the infected group were higher than those in the uninfected group on 1 day 3 days and 7 days after operation  $P<0.05$  serum miR 146a and Galectin 3 at 3 days postoperatively were positively correlated with CPIS score and APACHE score at 3 days postoperatively  $P<0.05$  at 3 days after operation the AUC value of miR 146a and Galectin 3 combined to diagnose pulmonary infection after PCI was greater than that of any indicator alone  $P<0.05$  serum miR 146a and Galectin 3 levels of effective patients were lower than those of ineffective patients on 1 day 3 days and 7 days after operation  $P<0.05$ . Conclusion The expression of serum miR 146a and Galectin 3 in patients with pulmonary infection after PCI is closely related to the CPIS and APACHE scores which can be used as an objective basis for clinical diagnosis. Moreover the effect of conventional anti infective therapy in patients with high expression is not good and the clinical need to adjust the treatment plan in combination with its expression in order to promote the outcome of the disease.

KEY WORDS Percutaneous coronary intervention Pulmonary infection miR 146a Galectin 3 Receiver operating curve

Percutaneous coronary intervention PCI

1

1.1

PCI

2019 1 2019 12

507 PCI

n=76 n=431

PCI

1

PCI

APACHE

CPIS

PCI

2

miRNA

PCI

P>0.05 1

miR 146a

3 Galectin 3

1.2

1.2.1

PCI

PCI

miR 146a

PCI

1 n % -

Table 1 Comparison of general information between 2 groups n % -

		n=76	n=431	t/ <sup>2</sup>	P
		58.64±5.31	57.65±5.11	1.188	0.235
		43 56.58	219 50.81	0.860	0.354
		33 43.42	212 49.19		
	kg/m <sup>2</sup>	21.03±0.85	20.96±0.83	0.676	0.500
		15 19.74	64 14.85	1.173	0.279
		12 15.79	51 11.83	0.929	0.335
		10 13.16	30 6.96	3.415	0.065
PCI	min	25.41±3.05	25.16±3.11	0.648	0.517
		44 57.89	256 59.40	0.060	0.806
		32 42.11	175 40.60		

miR 146a Galectin 3  
PCI  
2017 4 1 3 7 d miR 146a  
Galectin 3  
1.2.2  
miR 146a  
2 mL  
TRIzol  
PCR  
miR 146a Galectin 3  
2 mL 8 cm 3500r/  
min Galectin 3  
1.3  
146a Galectin 3 1 3 7 d miR  
7 d CPIS 1 3  
APACHE CPIS 5  
X 7 12  
6  
APACHE  
71 17  
miR 146a Galectin 3 CPIS APACHE

2.2 CPIS APACHE

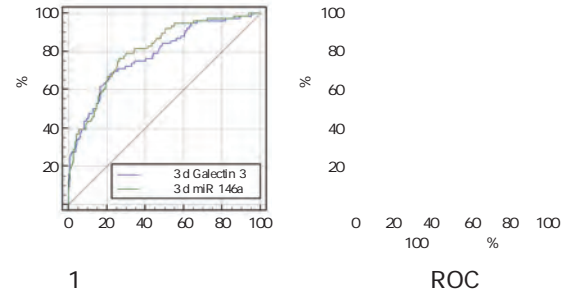
r>0.6  
 Galectin 3 3 d CPIS APACHE  
 P<0.05 3

3 CPIS APACHE  
 Table 3 Correlation between each serum index and CPIS, APACHE score

		miR 146a		Galectin 3	
		r	P	r	P
CPIS	1 d	0.481	<0.05	0.485	<0.05
	3 d	0.511	<0.05	0.451	<0.05
	7 d	0.504	<0.05	0.446	<0.05
APACHE	1 d	0.502	<0.05	0.474	<0.05
	3 d	0.499	<0.05	0.513	<0.05
	7 d	0.513	<0.05	0.462	<0.05

2.3 ROC

3 d miR 146a Galectin 3  
 PCI ROC  
 AUC > 3 d miR 146a  
 3 d Galectin 3 1 4



1 ROC  
 Figure 1 ROC curve of the single and combined diagnostic value of each index

4 ROC

Table 4 ROC analysis of the single and combined diagnostic value of each index

	AUC	95%CI		%	%
3 d miR 146a	0.805	0.768-0.839	>6.5	76.32	73.55
3 d Galectin 3	0.781	0.743-0.817	>25.18 ng/mL	68.42	77.96
	0.853	0.819-0.882	-	72.37	86.08

2.4

51 25  
 1 3 7 d miR 146a Galectin 3  
 P<0.05  
 3 d miR 146a Galectin 3  
 1 7 d P<0.05 5

5

Table 5 Serum expression of patients with different therapeutic effects

	n		1 d	3 d	7 d
miR 146a	51	3.95±1.28	5.58±1.67	7.05±2.11	4.67±1.41
	25	4.01±1.16	6.89±2.07	8.84±2.07	6.37±1.69
	F P		14.694	<0.001	
	F P		22.671	<0.001	
	F P		11.558	<0.001	
Galectin 3 ng/mL	51	14.96±4.49	19.67±5.90	24.96±6.44	15.58±5.03
	25	15.14±4.28	25.57±5.87	31.53±6.69	22.12±4.78
	F P		8.442	<0.001	
	F P		19.785	<0.001	
	F P		10.227	<0.001	

3

PCI 23.8%

CPIS APACHE

PCI

9

1 3 7 d miR 146a Galectin 3

miR 146a Galectin 3

Zhou Y <sup>10 11</sup>

3 d

7 d

Galec

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Galectin 3

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<sup>12</sup>

Galectin 3 PCI

1030

%

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PCI

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miR 146a

miR 146a

<sup>13</sup>

PCI

miR 146a

miR 146a

3 d

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miR 146a

miR 146a

<sup>14</sup>

PCI

miR 146a

miR 146a Galectin 3

ROC

3 d miR 146a Galectin 3

PCI

AUC

0.853

# EB

EBV EB

EBV EBV EBV EBV

EBV EBV EBV

## Progress of researches on Epstein Barr virus laboratory detection technology

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**ABSTRACT** Epstein Barr virus is a ubiquitous pathogen which is mainly transmitted by oral secretions and is closely related to a variety of malignant tumors. EBV detection methods mainly include serological and molecular biology methods each of which has advantages and disadvantages. Selection of the appropriate method depends on the clinical needs and multiple methods can be used in combination when necessary. To provide a more reliable basis and reference for the diagnosis and treatment of EBV related clinical conditions further evidence and consensus are needed to promote the standardization of EBV laboratory testing technology. This article introduces the current status of EBV detection technology and provides references for the diagnosis treatment and prognosis of EBV related diseases.

**KEY WORDS** EBV detection technology Antibody detection Nucleic acid testing

EBV epstein barr virus EBV 1964 encoded small RNA EBER 1 2 RNA  
 burkitt s lymphoma BL  
 1 EBV EBV  
 9 2  
 EBV EBV  
 3 T B  
 EBV 172 kb 85  
 DNA  
 6 EBV nuclear antigen EBNA EBV  
 3 latent membrane protein LMP 5 EBV hodgkin s  
 RNAs EBV RNA EBV lymphoma HL non Hodgkin

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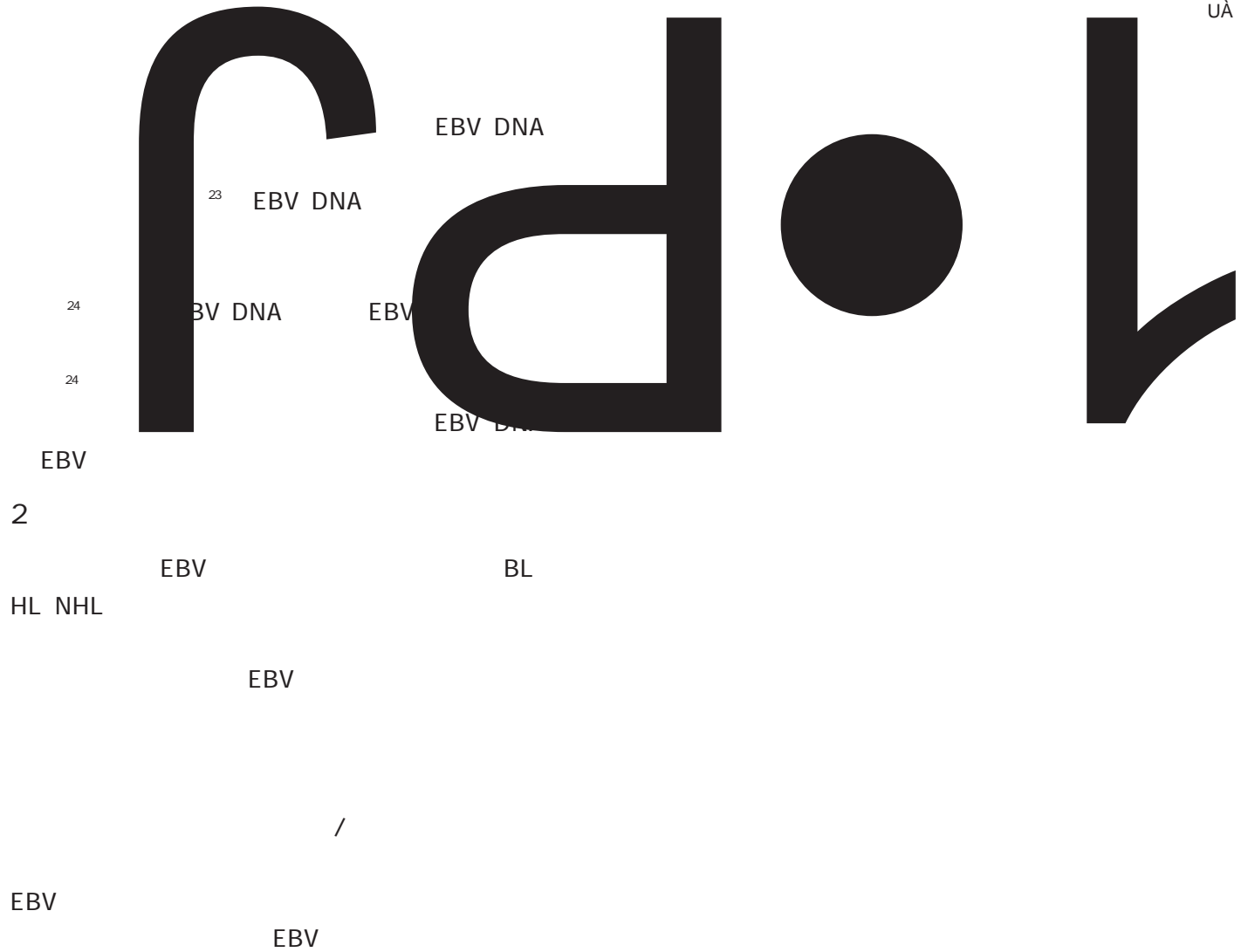


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