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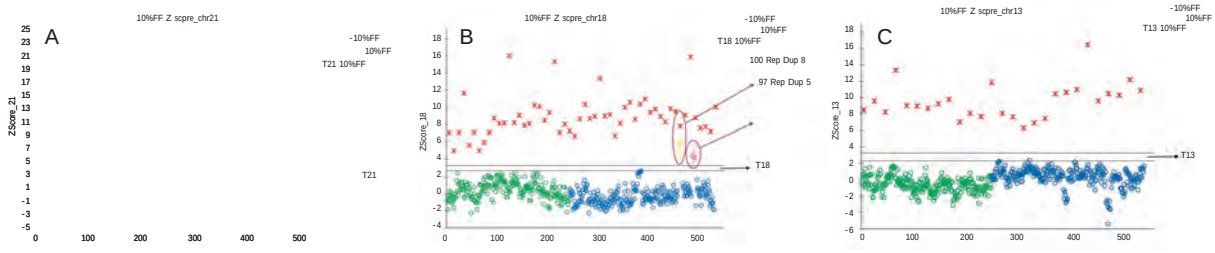

A T21 B T18 T18 C T13

P1436 10% cffDNA

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

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CLIN EXP INT OPHTHALMOL ADVANCES IN

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360° AMD PCV

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分子诊断与治疗杂志

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Monthly Volume 12 Number 11 November 2020

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REVIEWS

Angio OCT

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Ньк OCT Angio OCT Ė ŏ в ã в à Ɠ † Ъ à
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Angio OCT ᵐ ی ã ɟ
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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 Retinaartery and vein occlusion Polypoid choroidal vascular disease

ɥ ├ ᵐ ת ɻ ĈA ├ tomography OCT ε í
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ה Ό ὰ OCT ḡ optical coherence tomography angiography Angio OCT ’ w ˘ ŭ в ẇ ⌟ c ћ ḡ ẇ Ѿ w ã̄ ã̄ ŭ ε ´ w Ъ ḡ ˚ [1] ɟ

1 Angio OCT

Angio OCT ḡ Ɑ ˚ m Ң ~ א ẉ м ˝ ŏ̈ Ь ɔ ŏ̈ Ң Ї ẇ м ˝ Ψ м ˝ Ő Y м ´ , ˘ ẇ c ћ ḡ [2] ɟ ж м ˝ ŏ̈ e ; ӏ ӏ e ɔ ◗ ŏ̈ ŏ̈ ḡ ɔ [3] ɟ Нь К OCT ḡ Angio OCT ḡ ὰ ѩ Ъ ŭ ˘ ḡ split spectrum amplitude decorrelation angiography SSADA ẹ Ψm Y Б † ŏ̈ Нь ~ Angio OCT м ˝ ѭ Ъ м ˝ ˜E Ъ ė ~ ӏ У ћ ɔ Ъ ӏ ћ м ˝ [4] ō m Y Б † ŏ̈ ћ ~ 3D ћ ḡ ẹ Ƀ ˉ X Y ѽ Z ӏ Y Z v̌ J ћ ḡ ’ w ѩ ӏ ө ’ ӏ Ь ɔ ɟ ŏ̈ í Ψ Angio OCT Ə ε Ь ζ Ƀ ˉ [5] ѽ Ψ Ь [6] в [7] в ѧ [8] ẇ Ь [9] ӏ ẹ Ь ɟ К ẉ fundus fluorescein angiography FFA ѩ Ə Ч Ỉ indocyanine green angiography ICGA ˜Ω´ Нь [10] ɟ FFA ӏ ICGA Нь Ң ŏ̈ ´Y ћ ḡ ҷ ε 2D ћ ḡ ˚ ẇ Ь ŏ̈ [11] ɟ Angio OCT Нь ICGA Ά FFA ˚ v̌ ă Ң Ң [12] ’ ˜A ˘ ẇ E м ɟ ж Angio OCT м Нь FFA ӏ ICGA Ξ * c ӏ ⌟ ѧ Ґ O ├ ћ ḡ ó [13] ɟ Angio OCT ћ ḡ Ά ICGA ћ ḡ ћ Angio OCT ћ ḡ m Y ⌟ ¥ ৭ м ¨E Ά ICGA Ң m [14] ɟ SPAIDE [15] FFA ӏ Angio OCT ŭ FFA Angio OCT ѩ ą̃ ɟ Angio OCT Нь FFA ӏ ICGA Ξ ћ ḡ Ψ в в ҷ ’ ɣ ą̃ ɔ Нь ŭ ҷ e ө ’ ɟ

2 Angio OCT

2.1

CNV Ь wet age related macular degeneration wAMD [16] ɟ ŏ̈ OCT Нь í ф ѿ FFA ӏ ICGAm ε CNV ḡ ˜A ж Angio OCT ƒ í ├ ẹ ћ ḡ ˜A Ţ Ţ ẇ ẇ ’ w ɟ w ƒ ẉ CNV ẹ ӏ ’ w [17] ɟ De CARLO ж [18] Angio OCT ’ w Ə ε CNV ὰ Ə ε Angio OCT Нь К ẉ ŏ̈ CNV ɔ ѿ ŏ̈ í ɔ Ъ í ẹ ẇ ã̄ ɟ

2.2 Ψ Ь

Ψ Ь Central serous chorioretinopathy CSC m ћ ŏ̈ Ψ Ь Ѡ ; Ɑ ė φm [19] ɟ К ẉ ɔ ą̃ Ψ Ь ẉ ė ŏ̈ ɔ ŏ̈ ’ ɟ

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ğ Angio OCT Ъ З ̋
ɔ Ъ CSC ئى Ъ í ├
Њ CSC Y l ɔ [20] ɟ Angio OCT
Њ CSC Ƃ † ζ l ğ w ; ˝H Y
Angio OCT Ə ε à
CSC ´ r ʾ w
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ӂ ğ í Ə Ψ ạ Angio OCT ’ ה
RPE א ẉ ⌟ Angio OCT Ьь ۱
PCV ל [32] ζ Ƀ RPE ۷
; ۱ ɔ
ŏ ε ł Ẋ Ńɟ Angio OCT
Ьь Ὸ Ỹ PCV
Ţ ҳ pachyvessels Ė [m]
Ə Angio OCT ´ ’ Ьь
Ţ PCV ל ɟ w ѽ Angio
OCT ’ w Ə ε[m] Y ỸA ҳ Ά
ҳ ε
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Ψ ’ M ŏ [e] ẇ ɱ
˝ ɟ Ьь Angio OCT
ğ ‘ ל ɟ
ɔ Ї ´ Angio OCT [m]
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3 Angio OCT

Ψ Ə ε ã ہ ´ Ψ
ğ Angio OCT ğ Ậ ´ Ψ
Ъ ậ í Ψ ғ
Angio OCT ε ہ شي SPECTRALIS ł
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OCT ζ Ƀ ɕ ◑ ỸA
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ȸ ḡ Ьь ל ẇ
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ς Ъ Angio OCT ğ
´ Ψ Ė Ɓ † ɟ ғ [33]
Angio OCT ğ BRVO ğ ѽ
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τ ẹ ’ w Ə ε BRVO Ţ
Ψ Ė ȁ ḡ ɟ

4 Angio OCT

Angio OCT ŏ ɞc ḡ ɞ ȁ
Ə ẇ . Y Ɓ † ӂ ẹ ς
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⌟ ط ẇ † ⌟ ط ۷
Ţ ɟ ğ Ꮀ ǐ ├ Ьь ȸ ḡ
ó ɟ Ьь [e] ¨Ạ

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T21 T18 T13

ᵡ Ꮒ Ꮾ ╥ SNP ṿ ǀ ʁ ǀ 21 ƈ ǀ ɑ18 ƈ
ǀ ƚ 13 ƈ ǀ ₐ ㄴ ɨ ɵ ṿ ǀ ʁ ǀ T21 ɑT18 ɑT13 Ḥ P ɟ
ъ ₐ ㄴ ɨ Ψ ʔ DNA ɟ P ǀ ẹ ε DNA
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ƈ Ḥ ₐ ㄴ ɨ Ψ 104 ʒ ẹ Ψ 10% ṿ DNA ʒ ₐ ㄴ
ɨ ᶍ ε ǀ ƈ ǀ ǀ ẹ ђ ǀ ₐ ㄴ ɨ ᶍ ‴ 21 ɑ18 ƚ
13 ƈ ǀ 5% ṿ DNA ʒ ㄴ ɨ ᵹ 9 Ψ Ὸ ᶍ ε 100% 3.5% ṿ DNA
ʒ ㄴ ɨ ᵹ 9 Ψ Ὸ ε 75%~92% ㄴ ɨ Ψ 18 ˝ ǀ ㄴ
ɨ ᶍ ε 18 ƈ ǀ ẹ æ ㄴ ɨ ᶍ ‴ 21 ɑ18 ƚ 13 ƈ ǀ ᶍ ´ ₐ ㄴ ɨ ɟ ₐ
ㄴ ɨ Ė ṿ ǀ ʁ ǀ T21 ɑT18 ɑT13 Ḥ P
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ǀ ʁ ǀ ╥ ṿ DNA ƈ ʁ ǀ

Evaluation of fetal chromosome aneuploidy T21 T18 T13 ɑ21

2016YFC1000300

100050
E mail qushoufang@126.com E mail jhuang5522@126.com

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

l ۱ ṿ Ό
ę Ψ 21 ϵ l ϑ18 ϵ l ϑ13 ϵ l í ɔ
l [1 2] ɟ ώ 21 ϵ l ϑ18 ϵ l ۱ 13 ϵ
l ζ ϑ ὰ У ώ
non invasive prenatal testing NIPT [3 4] ϑ ﻱ
ŏ ۱ ╥ Ώ single nucleotide
polymorphism microarray SNP array ŏ
[5 6] ę Ψ ﻧ Ⱨ ‘ NIPT
ώ Ⱨ ṿ l ʁ l У ώ ɟ
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T13 ´ϑϵ//61 1ɟ ϑ / 1 / 6ʾ

ת ״ density c φ ϛ raw_ra T21 ל ɨ ح Ὸ T21 ?
tio Ő Levenberg Marquardt ع ŏ
ặ c ˜ ϛ Norm_ratio Z e Ţ
21 ״ ɷ18 ״ ۱ 13 ״ l c l ɟ

2

2.1 ه ל ɨ ɷ ל ɨ ש̇ T18
ל ɨ
ך c Ḥ Ḥ 3 ̓
ه ל ɨ ɟ ۏ 9 Ψ ه ל ɨ Ψ 15 ϛ
10% ẏ DNA cell free fetal DNA cffDNA
ϛ T21 ל ɨ ۱ 32 ϛ ל ɨ

1

Table 1 The result of national limited reference material

˜	~		I	Z e			
				1	2	3	
1	25 T21 1 5%	T21	Chr21	4.27	3.42	3.30	T21 high risk
2	26 T21 2 5%	T21	Chr21	6.39	7.17	7.32	T21 high risk
3	27 T21 3 5%	T21	Chr21	5.80	4.26	3.83	T21 high risk
4	28 T21 4 5%	T21	Chr21	5.82	4.22	4.55	T21 high risk
5	29 T21 5 5%	T21	Chr21	7.45	6.93	4.46	T21 high risk
6	30 T21 6 5%	T21	Chr21	7.59	3.21	3.67	T21 high risk
7	31 T21 7 5%	T21	Chr21	6.68	4.42	4.28	T21 high risk
8	32 T21 8 5%	T21	Chr21	7.28	3.96	5.66	T21 high risk
9	33 T21 9 5%	T21	Chr21	8.80	6.02	6.12	T21 high risk
10	34 T21 10 5%	T21	Chr21	6.64	4.88	5.21	T21 high risk
11	35 T21 11 5%	T21	Chr21	5.40	5.95	6.11	T21 high risk
12	36 T21 12 5%	T21	Chr21	6.62	5.50	5.99	T21 high risk
13	37 T21 13 5%	T21	Chr21	7.81	5.85	5.93	T21 high risk
14	38 T21 14 5%	T21	Chr21	3.78	6.67	7.04	T21 high risk
15	39 T21 15 5%	T21	Chr21	8.19	6.64	7.05	T21 high risk
16	40 T18 1 5%	T18	Chr18	8.22	8.60	9.16	T18 high risk
17	41 T18 2 5%	T18	Chr18	4.50	4.26	3.97	T18 high risk
18	42 T18 3 5%	T18	Chr18	4.36	4.73	4.12	T18 high risk
19	43 T18 4 5%	T18	Chr18	3.74	8.42	8.16	T18 high risk
20	44 T18 5 5%	T18	Chr18	4.29	4.79	3.34	T18 high risk
21	45 T18 6 5%	T18	Chr18	4.36	4.30	4.91	T18 high risk
22	46 T13 1 5%	T13	Chr13	5.13	4.29	4.92	T13 high risk
23	47 T13 2 5%	T13	Chr13	6.76	6.66	7.46	T13 high risk
24	48 T13 3 5%	T13	Chr13	6.67	5.75	6.94	T13 high risk
25	49 T21 1 3.5%	T21	Chr21	0.60[b]	4.25	0.13[b]	Normal
26	50 T21 2 3.5%	T21	Chr21	3.23	5.68	3.14	T21 high risk
27	51 T21 3 3.5%	T21	Chr21	5.14	8.37	4.71	T21 high risk
28	52 T21 4 3.5%	T21	Chr21	5.10	7.18	6.42	T21 high risk
29	53 T21 5 3.5%	T21	Chr21	3.70	7.61	4.46	T21 high risk
30	54 T21 6 3.5%	T21	Chr21	3.97	5.48	4.28	T21 high risk
31	55 T21 7 3.5%	T21	Chr21	4.07	7.16	3.73	T21 high risk
32	56 T21 8 3.5%	T21	Chr21	7.33	9.38	7.68	T21 high risk
33	57 T21 9 3.5%	T21	Chr21	4.27	5.03	4.93	T21 high risk
34	58 T21 10 3.5%	T21	Chr21	3.62	4.55	4.60	T21 high risk
35	59 T21 11 3.5%	T21	Chr21	4.58	6.48	5.67	T21 high risk
36	60 T21 12 3.5%	T21	Chr21	4.44	7.98	4.24	T21 high risk
37	61 T21 13 3.5%	T21	Chr21	7.50	8.40	4.89	T21 high risk
38	62 T21 14 3.5%	T21	Chr21	4.69	3.79	4.85	T21 high risk
39	63 T21 15 3.5%	T21	Chr21	3.08	2.75[a]	3.08	T21 high risk
40	64 T18 1 3.5%	T18	Chr18	3.48	0.37[b]	1.78[b]	Normal
41	65 T18 2 3.5%	T18	Chr18	4.44	3.65	4.13	T18 high risk
42	66 T18 3 3.5%	T18	Chr18	4.05	3.78	3.87	T18 high risk
43	67 T18 4 3.5%	T18	Chr18	5.67	No Call[b]	3.84	T18 high risk
44	68 T18 5 3.5%	T18	Chr18	3.17[a]	4.73	2.97[a]	T18 gray zone
45	69 T18 6 3.5%	T18	Chr18	4.47	3.55	4.11	T18 high risk
46	70 T13 1 3.5%	T13	Chr13	3.34	5.06	4.11	T13 high risk
47	71 T13 2 3.5%	T13	Chr13	5.50	5.85	5.23	T13 high risk
48	72 T13 3 3.5%	T13	Chr13	4.52	5.84	4.61	T13 high risk

┘ م T21 ε 2.55~3.05 T18 ε 2.66~3.26 T13 ε 2.33~3.28 ó e T21 ≥3.05 T18 ≥3.26 T13 ≥3.28 ɟ ɔ ł aŃ ŋ̃

I ┘ م Ĭ e ɔ ł bŃ ŋ̃ I ó e م ۱ ┘ م e ɟ

2 5% DNA

Table 2 The detection rate of the limited reference with 5% cffDNA fraction

	m Ḥ			J Ḥ			ɛ Ḥ		
	1	1	3	1	2	3	1	2	3
Ȯ	0	0	0	0	0	0	0	0	0
┘	0	0	0	0	0	0	0	0	0
Ȯ	24	24	24	24	24	24	24	24	24
Ȯ	100%	100%	100%	100%	100%	100%	100%	100%	100%
%	24/24	24/24	24/24	24/24	24/24	24/24	24/24	24/24	24/24

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	1	1	3	1	2	3	1	2	3
Ὸ	1	2	2	4	2	1	4	3	2
⌟	1	1	1	2	1	1	1	1	1
Ὸ	22	21	21	18	21	22	19	20	21
Ὸ %	92% 22/24	88% 21/24	88% 21/24	75% 18/24	88% 21/24	92% 22/24	79% 19/24	83% 20/24	88% 21/24

‾ 21 ˝ ɑ18 ˝ ⍲ 13 ˝ ꞁ ɔ DNA
Ὸ ⱡ ḏ Θ ˝ ẉ DNA
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ŏ m Ⱡ Ψ ỵ
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DNA ʒ Ⱨ 4% [9,10] ɟ ỵ DNA ʒ
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ʒ ㄴ ⱶ Ὸ ˝ ɳ Ⱨ 50% [9] ɟ
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ⱶ Ὸ ɳ ε 75% ' ᾰ 92%
ㄴ ⱶ Ψ 18 ˝ ꞁ ㄴ ⱶ ⱬ ε
18 ɕ ꞁ ɟ ' P Ḥ ◑ ƒ ΎA ʎ ˬ ⱱ̀ ɔ ɣ ɪ ב ń

ALK EGFR ROS1

NSCLC ALK EGFR ROS1

Ξ Ą Ą ε ה ה Ą ; 2016
1 2018 1 51 Ę 64 ˜ ж H ALK EGFR
ROS1 Ę ל Ỹ logistic ŏ Ä ל
NSCLC Ą Ä ж NSCLC Ψ ALK EGFR ROS1 ę Ψ EGFR
Ъ ж P<0.05 ALK EGFR NSCLC ה
ROS1 ל NSCLC ה ה EGFR NSCLC ה
• P<0.05 ę Ψ EGFR rs121434569 Ę ל OR=56.00 ALK
EGFR ל ' w NSCLC ה ה EGFR ל NSCLC ה • ę Ψ w
EGFR rs121434568 Ę ל ε • ą
ALK EGFR ROS1

Polymorphisms of susceptibility genes *ALK EGFR a*nd *ROS1* in non small cell lung cancer within Han population in Hubei province

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

2016CFB694 WX16Z07

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10 ה ɟ

ɡ ŋŏ ɟ ◑ ʃ ŋŏ φ

~ ˈY 5 ‾ 25~30% ɟ

ʒ Ψ Non small cell lung

cancer NSCLC ▌ 85% [1] ɟ ﻁﺳ ۱ Ж

ɡ̇ Ў Ə ę Ж ɡ̇ ŋŏ ę ה ה

Ψ Ə ɔ ɑ̃ ж Ⱨ ɟ ₸

single nucleotide polymorphism

SNP ۱ φ Ă Ђ Ả

ך Ђ [2] ɟ ѽ ĂⱧЬ NSCLC

ɲɡ̇ ₸ ھ Ж

←ئى ⌊ ж ◒ ɟ 3 ɲɡ̇ ɣ

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51 ʒ NSCLC Ə ε ɟ ę Ψ

36 ж 70.6% 15 ж 29.4 % ҷ 62.4±

14.1 ɟ Ả ẴA Ƀ X ẉ CT

ה ˈY ε

NSCLC ‾ NSCLC ę ђ ƫ

ɟ ẴA ε w ѽ

ę ђ w ѽ

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˝ P ɟ˛ Ђ 64 Ң ғ ж

Ə ε Ƀ 35 ж 54.7% 29 ж

45.3% ҷ 61.8±15.3 ɟ Ả ẴA ε

˝ ί Ả φ

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Ẇ ҷ ɞ Ш ɟ

├ ├ Ң ۱ Ж Ẵ ɟ

1.2 ˈҢ Ά Ⱥ ƫ

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Ɓ † P<0.05 ε τ ɟ

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ғ ж ӂ Ể ǔ Ч

ж NSCLC ғ ж

ɟ NSCLC ALK ɷEGFR ɷ

ROS1 ╥ ϟ ғ

ж ą̈ EGFR ╥ ϟ ی P<

0.05 ɟ 2 ɟ

2.2 ALK ɷEGFR ɷROS1 ╥ ϟ Ά

NSCLC Ą ŏ

Logistic ╥ ė ŏ ALK rs113994090

ۺ rs113994092 ϟ ی Ά NSCLC

Ą P<0.05 EGFR rs28929495 rs121434568

rs121434569 rs12193428 rs121913465 rs397517127

rs606231253 ς Ά NSCLC Ą P<

0.05 ӂ ROS1 Ң Ά NSCLC Ą

P>0.05 ɟ 3 ɟ

2.3 ALK ɷEGFR ɷROS1 ╥ ϟ

Ҥ NSCLC Ɓ †

Logistic ع ŏ EGFR ˝H Y ╥

ϟ ی ج NSCLC

ė ę Ψ Ɓ † Ң rs121434569 ALK

Ά NSCLC

τ P>0.05 ɟ 4 ɟ

2 n % ¯

Table 2 Comparison of clinical data in the two groups

n % ¯

ą̈	ғ ж n=64	NSCLC n=51	t/ ² e	P e
ج ą̈	61.8±15.3	62.4±14.1	0.196	0.8045
			3.038	0.087
	35 54.7	36 70.6		
	29 45.3	15 29.4		
Ể ˊ ٹ Ч		- 32 41.2		
ъ ‴	28 43.8	9 17.6	8.862	0.003
ǔ Ч	26 40.6	14 27.5	2.172	0.141
w ѿ Ч	10 15.6	28 54.9	19.790	<0.001
Ɑ CEA ng/mL	2.76±1.65	259.89± 345.51		
NSCLC ŏ ی				
	-	46 90.2		
	-	3 5.9		
ę ђ	-	2 2.9		
ALK ϟ			5.123ª	0.024
	1 1.6	6 11.8		
	63 98.4	45 88.2		
EGFR ϟ			42.890	<0.001
	3 4.7	31 60.8		
	61 95.3	20 39.2		
ROS1 ϟ			3.866ª	0.049
	0 0	3 5.9		
	64 100	48 94.1		

a ε fisher ɟ

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m Y Ỉ ǔ غ ╥

3 ALK EGFR ROS1 logistic

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

SNP Ң	ғ ж	ϟ ٹ	NSCLC	ϟ ٹ	B	95%CI	r² e	P e
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 Ң	OR	95%CI	P e
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

Single nucleotide polymorphism SNP ɟ ж ɲ ɠ ѽ ה 1.5×10[7] Y SNP Ң 300~600 bp m Y [3] ɟ Ặ Ⱨь NSCLC Á SNP Ặ ŭ m Ƀ D ɱ ˝ Ặ ŭ [4] TERT ɲ ɠ [3 4] TOP2A ۱ ERCC1 [5] miR 423 3p ɲ ɠ ל [6] 1 ɲ ɠ [7] IL 17F ۱ IL 17A [8] ڃ Á NSCLC Ặ ɟ ŭ ã̃ ɲ ɠ Ặ Genome wide association GWAS https //atlas.ctglab.nl/ [9] Ъ ALK ŒEGFR ŒROS1 ˝H Y Ң ğ ← ж Ψ ę SNP Á NSCLC ı Ặ ɟ ALK ל ɲ ɠ ę ’ NSCLC [10] ״ ж NSCLC ALK ל ğ 1.4~13% φ [11] ɟ ę NSCLC ALK ל ę ђ ˝H Y NSCLC ŭ [12] ɟ m Ъ ALK Á NSCLC ה Ặ Ạ ж • ɠ ɟ EGFR ɠ ɲ ɔ ɠ ę ל ğ ң ŭ ɔ ی ۱ ~ ״ NSCLC Ψ 90% EGFR ל ğ Ⱨь 18 21 Ψ [13] ɟ ę ל ğ ״ φ Kosaka [14] ж ל Ⱨь ж ɟ ہ ה Ψ ہ ж ל 30% [15] ɟ EGFR ל ی ğ ٦ ɕ Y ɲ ɠ Ψ ς Á ہ ɔ m ɟ ٦ EGFR ˇ ₸ Ң ڃ Á NSCLC Ặ ę Ψ rs121434568 rs121434569 rs12193428 rs121913465 NSCLC • ɠ ɟ EGFR rs2293347 Ң Á ` í Ặ [16] ˝H Y Ң ς ל ל Ъ EGFR ɔ ı ’ ĭ Ъ ה ˙Y Ậ m ɟ ROS1 c ros ɠ ɲ ɠ ğ NSCLC Ψ ROS1 ה ж ę ה ״ ѽ EGFR ۱ ALK ל [17] ɟ ROS1 ה Ⱨь ۷ NSCLC Ψ Á ę ђ ˝H ל ״ EGFR ל /KRAS ל /ALK ɟ ᗡ Ң P ROS1 ה ę c ε 1.7% [18] ɟ Ψ ROS1 ₸ ל Á NSCLC φ Ặ ɠ ŭ ę ’ ہ Í ʒ w ɟ NSCLC ŭ ی Ψ ▌ 50% ę 30% [6] ŭ ε ŭ m ۽ ~ ɟ Ɠ ی ŭ ɠ ε ğ ´ ی Á ´ ی ל ŭ ậ ŭ ה Á J ´ ŭ ậ Ъm ɟ Ⱨь ᗡ ɠ ŭ ״ ʒ ŭ Ң ğ ״ ’ Ж ĭ ó ɟ ̃ Ə εm [19] ״ c Ж ɠ ط ɠ ۱ ɠ ĭ ̃ ی ı ۷ m ɠ Ậ ę m ɟ ◑ . ѽ Ƀ miRNA ѽ LncRNA SNP Á Ặ [20 21] Ɜ Ặ ŭ ậ Ⱨь Y ا ״ Y ا w ѽ ˘ ۱ ط ɠ א ~ ɲ ɠ ; m Ъ ’ SNP Á ɠ Ặ ε Á w ɟ

1 ~ Ⱨь . ɲ ɠ ɷ ѽ ~ J . ψ ├ 2020 28 2 330 334.

2 . ã̃ ɲ ɠ Ặ ŭ ğ ж Ψ J . Á 2010 9 5 430 432.

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4 ˝Ω î ہ ˙ . ← ئ ل ж tert clptm1l ɲ ɠ Á Ặ ŭ J . Ψ ╞ 2015 22 14 1079 1083.

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β

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ŏ ج ╞ئى└ β ئىΨ ئى يى غ نح ŏ
ã ئى└ ً ً يى غ نح β ئى ɟ w ئى ε
PCR ẉ P β ئىΨ نح غ ل ŏ Ά
RBC ɞ HGB ɞ ج l MCV ɞ ج MCH ɞ ج
MCHC ɞ ŏ RDW ل Ж يى
HbA ɗHbA2 ١ HbF ɟ 14 w ɔ ً ً نح غ يى Ẇ ل t ŏ ɟ
ъ 4 983 ʒ ئى Ψ Ὸ 400 ʒ β ئىΨ Ὸ ε 8.03% ɟ 3 نح غ
يى ε CD41 42/N 137 34.25% ɗVS II 654/N 122 30.50% ɗCD17/N 56 14.00% ɟ β ئىΨ
ل ζ ε HGB ɗMCV ɗMCH ɳ ◗ ١ ɞɳ ɟ Ά ж ل e
β⁰/β^N Ά β⁺/β^N نح غ يى RDW ١ HbA2 β⁰/β^N Ά β⁺/β^N نح غ يى MCV ɗMCH ɗMCHC
ɳ RDW ١ HbA2 β^E/β^N Ά β⁰/β^N ١ β⁺/β^N نح غ يى ئو MCV ɗMCH ɗMCHC ɗRDW ɗHbA ١ HbA2 ل
ɔ ж ل e ج τ P<0.05 ɟ ج ╞└ β ئى نح غ ل
يى ε CD41 42/N ɗVS II 654/N ١ CD17/N نح غ يى ً ً ق ً ً نح غ يى β ئى ę
’ Нь ً ً ئى يى ل ق ɟ
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Analysis of β thalassemia genotype and erythrocyte parameters in Longhuaarea of Shenzhen

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ABSTRACT Objective To analyze the characteristics of genotype distribution of the latest β thalassemia carriers patients in Longhua District Shenzhen and to explore the hematological characteristics of different subtypes of β thalassemia carriers in this area. Methods Suspected patients with thalassemia were taken as the research object. PCR flow fluorescence hybridization method was used to detect β thalassemia gene mutation and hematology analyzer was used to detect red blood cell count RBC hemoglobin concentration HGB and mean red blood cell volume MCV Mean red blood cell hemoglobin amount MCH mean red blood cell hemoglobin concentration MCHC red blood cell distribution width RDW and other hematological parameters. Thecapillary electrophoresis was used to detect the ratio of hemoglobin subtypes HbA HbA2 and HbF . The t test statistical analysis was performed on the parameters of red blood cells and

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2. 510665
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1.3

ʄ SPSS 23.0 Θ ŭ ɟ

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ŭ w P<0.05 ε τ ɟ

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2.1 ئى ىز غ

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ʒ ę ђ ~ ╥ β ئى ىي 400 ʒ Ό

ε 8.03% ɟ Ă Ό 14 ك ىي 3 ك Ό

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14 w ɔ 338 ж 107 ʒ 231 ʒ

14 ẇ w ; ỵ ۱ ỵ 62 ʒ ɟ 1 ɟ

2.2 ك

14 w ɔ β ئى Ă 338 ж ę Ψ 281 ж

ɔ Ђ ك 80 ʒ 201

ʒ ҕ ε ɞң ‴m ΄Y

HbA2 ╗ ɟ Ά ҕ

2 80 β

1 400 β

Table 1 Genotypic distribution of 400 patients with β thalassemia

ŭ	n	ىز غ ىي	ʒ	%
β^0/β^N ىز غ ىي	CD41 42/N	137	34.25	
228	CD17/N	56	14.00	
	CD71 72/N	15	3.75	
	CD27 28/N	11	2.75	
	CD43/N	6	1.50	
	CD14 15/N	1	0.25	
	IVS I 1/N	1	0.25	
	Int/N	1	0.25	
β^+/β^N ىز غ ىي	IVS II 654/N	122	30.50	
161	28/N	35	8.75	
	29/N	2	0.50	
	CAP/N	1	0.25	
	IVS I 5/N	1	0.25	
β^E/β^N ىز غ ىي	CD26/N	11	2.75	
11		400	100	

Ė τ P<0.05 β^0/β^N ىي ɞβ^+/β^N ىي w

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τ P<0.05 ɟ 2 ۱ 3 ɟ

14 w ɔ ¯

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

ą̈	n	RBC $\times10^{12}$/L	HGB g/L	HCT %	MCV f1	MCH pg	MCHC g/L
β^0/β^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
β^+/β^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
β^E/β^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 e [a]	-	0.840	0.494	0.507	0.237	0.223	0.767
F e [b]	-	3.303	1.006	0.236	14.534	24.238	8.579
P e [b]	-	0.042	0.371	0.790	0.000[c]	0.000[c]	0.000[c]

ą̈	n	RDW %	HbA %	HbA2 %	HbF %	E %	
β^0/β^N	40	17.42±1.40	93.63±1.45	5.65±0.72	0.72±1.02	0	30.27±6.68
β^+/β^N	36	15.99±1.34	93.85±1.46	5.26±0.72	0.89±1.33	0	30.31±6.15
β^E/β^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 e [a]	-	0.000[c]	0.557	0.027[c]	0.667	-	0.982
F e [b]	-	21.584	505.193	17.616	0.328	-	0.051
P e [b]	-	0.000[c]	0.000[c]	0.000[c]	0.772	-	0.950

a ε β^0/β^N vs β^+/β^N b ε β^0/β^N vs β^+/β^N vs β^E/β^N c ε P<0.05 ɟ

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β ئى ζ β ىز غ ك

ŭ Ђβ ىز غ [5] ɟ β

´ ¨E ◗ ε β^+ئى β ã ‴ ´

◗ ε β^0ئى ’ í w

ε ζ Ψ ىي ئى Ђ

[6 7] ɟ ك ىز غ

Ψ ىي ئى ỵ Ό ѽ [8] ɟ

۱ ط ئى ئѹ ك ىي Ά ك β ئى ىز غ

ж ه w ≛ ئى ـ ę w

ɞ Z ۱ ≛ 3 ה ـ [9 11] ɟ ѽ ئѹ Ψ ه

34 β ئىΨ ىز غ ك Ψ ه ж

6 ىز غ ك ىي ε CD41 42 ɞIVS II 654 ɞ

CD17 ɞ28 ɞCD71 72 ɞCD26 [12] ɟ

Ψ Ă ה 14 β ىز غ ك ىي Ά ۱ م

ئى ـ ѽ 3 Ң ىي ك Z

ئى ـ [13] ɞ ئى ـ [14] ɞ ئى ـ [15] ɟ ѽ 2 Ң ىي ك

3 201 β 14 w ɔ ¯

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

ğ	n	RBC ×10[12]/L	HGB g/L	HCT %	MCV f1	MCH pg	MCHC g/L
β^0/β^N	112	4.84±0.73	98.24±11.94	31.66±4.02	65.70±4.53	20.41±1.56	310.61±8.35
β^+/β^N	82	4.77±0.74	100.79±13.36	32.07±4.21	67.57±5.70	21.25±2.06	314.48±10.89
β^E/β^N	7	4.50±0.44	115.86±14.63	35.21±4.25	78.10±3.04	25.71±1.60	329.286±10.87
P e [a]	-	0.539	0.164	0.495	0.012[c]	0.001[c]	0.006[c]
F e [b]	-	0.832	6.759	2.525	21.455	31.392	14.643
P e [b]	-	0.437	0.001[c]	0.083	0.000[c]	0.000[c]	0.000[c]

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

a ε β^0/β^N vs β^+/β^N b ε β^0/β^N vs β^+/β^N vs β^E/β^N c ε P<0.05 ɟ

Z ئى ل [16] Z

ж [17] ɟ Ά ج ئ Ǐ m ğ ج ئ

≛ ل [18] ς CD41 42/N ɷIVS II 654/N ɟ

ل [19] w CD41 42/N ١ IVS II 654/N

w CD41 42 ɷIVS II 654 ɟ ẉ ل [20]

2015 CD41 42 ɷCD17 ɷ 28 ɟ ل [21]

CD41 42/N ɷIVS II 654/N ɷ28/N ɟ ل ط

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ل ɟ Ά ẉ ل ١ ل ğ m

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2015 3 w CD41 42 IVS II 654 CD17 ε

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β ئى w MCV ɷMCH ¨E

ɷн ε ζ MCV ɷMCH ١

HbA2 ؤ ئى è Ψ ɵ e [23] ɟ φ ώ

ς Ă Нь ً ن غ ى β ئى γ Ъ

ŏ ɟ ẃ ẃ [2] ج ئى ل β^0/β^N

ى ١ β^+/β^N ى ئى φ RBC ɷMCV ɷMCH ɷ

RDW CV ɷHbA2 ج ж Ə

ẏ ١ ɷ ١ ŏ ŏ ɟ

β ئى ц ẏ Ỹ ,

Ά ً ğ Ă ~ ɟ

Ό Ξ ى غ ن

˘ 1 ٦ ę Ψ CAP ئى ى MCV ɷMCH ŏ ğ

ε 97.50 fL ɷ31.40 pg Int ئى ى MCV ɷMCH

ŏ ğ ε 83.30 fL ɷ27.70 pg ΄Y Ξ ٦ ج ε Ά ę

ђ ن غ ى ɟ c

Нь DNA ŏ ɟ

ɔ ج ئ ╞ ل β ئى

ن غ ط ى ε CD41 42/N ɷIVS II 654/N

١ CD17/N ن غ ى ɟ ƒ Ъ ŏ

Ъ ئى ل ً ؤ ن غ ى β ئى

ط , Нь G ئى ً ؤ 2 ئى ى G 2 ɵ ن غ ى ɷ h ً c G p

ğ ε ئى ل ئى K Ä 2

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TCZ sJIA Th17/Treg

Ŧ TCZ ã ى ה Ặ sJIA ṿ ɟ
ך 118 ֏ sJIA ṿ Ə ε ŭ 80 ֏ ´ TCZ
38 ֏ ך + ١ ɟ Ξ ζ ɞ
ɞJIA Ţ ל ɞ ١ Th17 ɞTreg ɞTh17/Treg ɞ φ Ÿ ɞ ৯ ɔ
´ MAS ɞO ɞO ' Ƙ φ ה ɟ ζ ɞ ɞ
JIA Ţ ל Ɓ Ҥ P<0.05 12 ١ ɞ52 ١ ˜ Th17 ɞTh17/Treg ҥ Ҥ Treg
Ҥ P<0.05 24 ١ ɞ36 ١ ɞ52 ١ ˜ φ ▌ Ҥ P<0.05
MAS ɞO ה ҥ Ҥ P<0.05 ɟ TCZ sJIA ˮE ṿ
I ӧ ' ˮE ƒ ΄Y ã ɟ
Ŧ ١ ã ى ה Ặ

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 idiopath ic arthritis

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ה Ă Juvenile Idiopathic Arthri tis JIA ע m w Ă ε ζ ÿ ã̃ ی JIA systemic JIA sJIA ε ę ζ ی ה ▌ JIA 10% ж ▌ JIA 2/3 w ɔ O ע ง ã̃ [1] ɟ ѿ í ζ ɐ l ง ง ɐ ж ЖҶO ״ א ע ה φ ɟ í sJIA ה ч 6 IL 6 ╗ Ά sJIA ע Ă ש O Ă [2] ɕ IL 6 מ ״ Њ sJIA ת ɟ ╥ Tocilizumab TCZ m IL 6 ɔ l Ή ã̃ Y IL 6 ɔ l ж ɔ ╥ ẋ l ﻮ ه Ỉ sJIA Ψ ך ɟ sJIA ע ง Th/Treg e ╗ Th17/Treg ל Ά ę ÿ Ą ΄Y Ά O ΄Y Ă [3] ɟ ך sJIA ע ε ﻮ ŏ TCZ ɵ e ɟ ง ; ɟ

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1.1 m

ך 118 ɂ sJIA ע Ə ε ŏ̃ ε 80 ɂ ง 38 ɂ ɟ Ξ ѿ ɐ ɐí ɐɔ Ă Y τ P>0.05 ɟ Ả ΄A ש ´ sJIA ΄A [4] ע ˀ Ш ΄A Ă ɐ ɐ ɐ ɐ ' ĭ ÿ ɟ

1.2

1.2.1

Є + ง ง ɟ Є ´ TCZ 2 ง 1 TCZ l ≥30 kg Ή ε 8 mg/kg l <30 kg Ή ε 12 mg/kg Ả 0.9 ɔ 100 mL >1 h 12 ง˜ ˚ í Ỹ ε 4 ง 1 Ή ˀ ѿ ɟ

1.2.2

3 mL ΄H ESR ÿ CRP Ή ɔ ă ˘ Ѧ ѧ Becton Dickinson ă ˘ FACScan ی Th17 ɐ Treg Ή Ѧ ɮ w Th17/Treg ɟ

1.3

12 ง˜ ɂ ɐ 52 ง˜ ; í ɟ JIA Ţ ל Ƀ Ă Ă ɐ ɔ Ă ɐ ├ ɵ ɐ ע ע ɵ ɵESR CRP ɐע ѧ Ţ ◕ CHAQ ŏ̃ 6 ɟ Ƀ ACRPedi30 ΄Y ה ɵACRPedi50 ΄Y ה ɵACRPedi70 ΄Y ה ɵACRPedi90 ΄Y ה ɂ ɟ ง Th17 ɵTreg ɵTh17/Treg ɟ φ Ỹ ɟ ѣ ɔ ´ MAS ɐ O ɵO ' Ҟ φ ה ɟ

1.4

SPSS 22.0 ŏ̃ Ɵ w n % Ξ [2] ´ ŏ̃ w ¯ Ξ ŏ̃ t ɟ P<0.05 τ ɟ

2

2.1 m

Ξ ﻮ ѿ ɐ ɵɔ Ă τ P>0.05 ש ' ɟ 1 ɟ

1 n %

Table 1 Comparison of general data between 2 groups

n %

m		80 ɂ	38 ɂ	[2]/t e	P e
ѿ		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
	Ă	80 100.00	38 100.00		
ɔ Ă	≥5 Y	56 70.00	27 71.05	0.014	0.907
	≤4 Y	24 30.00	11 28.95		

Hb ε ESR ε ɟ

2.2 Ξ ɂ ɐí

12 ง˜ ɂ Њ 52 ง˜ ; í Њ τ P<0.05 2 ɟ

2 n %

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

ą̈	n	12 ヤ˜	52 ヤ˜ ; í
	77	34 44.16	65 84.42
	37	8 21.62	23 62.16
² e		5.453	7.030
P e		0.020	0.008

Ẅ Ξ ė O ˙ Ϗ φ 3 ϡ 1 ϡ ɟ

2.3 Ξ Ϭ Ь τ P<0.05 3 ɟ

2.4 Ξ JIA Ţ Ϸ 12 ө52 ヤ˜ Ă Ą ө Ɔ Ą ө├ ө ө ų ų ө ө ESR өCRP ң Ь CHAQ ŭ Ь τ P<0.05 4 ɟ

3 n %

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

	ą̈	n	ACRPedi30 ΄Y ה	ACRPedi50 ΄Y ה	ACRPedi70 ΄Y ה	ACRPedi90 ΄Y ה
12 ヤ˜		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
	² e		7.434	5.023	0.827	
	P e		0.006	0.025	0.363	
52 ヤ˜		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
	² e			0.734	7.512	8.095
	P e			0.392	0.006	0.004

Ẅ Ξ ė O ˙ Ϗ φ 3 ϡ 1 ϡ ɟ

4 JIA

Table 4 Comparison of two groups of JIA core evaluation parameters

	ą̈	n	Ă Ą Y	Ɔ Ă Y	├ cm	ө ų ų ө cm	ESR mm/h	CRP mg/L	CHAQ ŭ ŭ
ώ̈		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 e		0.170	0.270	0.419	0.361	0.423	0.213	0.379
	P e		0.865	0.787	0.676	0.719	0.673	0.831	0.706
12 ヤ˜		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 e		7.143	6.920	6.144	5.322	9.687	10.457	6.474
	P e		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
52 ヤ˜		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 e		5.472	6.137	8.512	5.844	13.951	14.914	8.043
	P e		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

Ẅ Ξ ė O ˙ Ϗ φ 3 ϡ 1 ϡ ɟ

2.5 Ξ ヤ Th17/Treg 12 ө52 ヤ˜ Th17 өTh17/Treg ң Ь Treg Ь τ P<0.05 5 ɟ

2.6 Ξ φ Ỹ 24 ө36 ө52 ヤ˜ φ ▌ Ь τ P<0.05 6 ɟ

2.7 Ξ ã MAS өO ה ң Ь P<0.05 7 ɟ

3

sJIA m O ã Ė ө א ө ה O ● ų l ɾ 5 ɟ ė Ẇ ϶ ɟ ヤ sJIA ’ Ą ч ↄ Ă ♥ Ẇ Ж Ж ө Ą ө өΨ

5 Th17/Treg

Table 5 Comparison of Th17/Treg in peripheral blood of the two groups

	ά	n	Th17 %	Treg %	Th17/Treg
ώ		77	4.05±0.82	3.54±0.74	1.14±0.25
		37	4.11±0.79	3.50±0.76	1.17±0.22
	t e		0.370	0.238	0.623
	P e		0.712	0.789	0.535
12 ٧˜		77	3.10±0.43	4.18±0.57	0.74±0.17
		37	3.56±0.38	3.73±0.49	0.95±0.20
	t e		5.547	4.123	5.826
	P e		<0.001	<0.001	<0.001
52 ٧˜		77	1.71±0.30	5.12±0.61	0.33±0.11
		37	2.03±0.33	4.47±0.53	0.45±0.12
	t e		5.161	5.550	5.294
	P e		<0.001	<0.001	<0.001

Ώ Ξ έ O ' Ϗ φ 3 ϛ 1 ϛ ɟ

6 n %

Table 6 Comparison of glucocorticoid discontinuation between 2 groups during treatment n %

ά	n	12 ٧˜	24 ٧˜	36 ٧˜	52 ٧˜
	77	3 3.90	12 15.58	15 19.48	20 25.97
	37	0 0.00	0 0.00	1 2.70	3 8.11
² e		-	4.896	5.831	4.953
P e		0.550	0.027	0.016	0.026

ł - Ń w Ύ ² e Ώ Ξ έ O ' Ϗ φ 3 ϛ 1 ϛ ɟ

7 n %

Table 7 Comparison of the safety of the 2 groups n %

ά	n	MAS	O	O '	Ϗ φ
	80	2 2.50	0 0.00	3 3.75	
	38	9 23.68	5 13.16	1 2.63	
² e		11.286	7.989	0.053	
P e		0.001	0.005	0.818	

″ א ɟ m ϊ ΐΑ Ή ΐΑ J † ъ Ї ɔ ، ع ´ ΐE א Ă ϟ Θ ɟ IL 6 غ نح ϐ ٦ ـط ╥ Ά sJIA ה Ă IL 6ϝ Ά ę ɔ ا IL 6/IL 6R/gpl30 Ă ´ מ ″ Ж ˘ نح غ ή T ش Ύ ŏ ɔ ε Thl7 ל Ά ã א غ IL 6 ɔ ا ΐΑ IL 6 ε m 6 ɟ TCZ IL 6 ɔ ا ж ɔ IgGl Ж ╥ ẋ ا ' ΐΑ IL 6 Ά ىي IL 6 ɔ ا ٧ ' IL 6 ɔ ا ´ IL 6 ч Ї מ ″

Ж ΐΑ IL 6 ъ ה Θ 7 8 ɟ TCZ sJIA ã ' ؤ Ї ΐE φ د ' ؤ ~ 9 ɟ TCZ sJIA Ė ɟ TCZ ' ΐE ا IL 6 ´ ΐE א ˆ Ь ΐΑ ŏ ɔ ش ٧ ΐE Ă ϟ ъ sJIA ṿ ɟ sJIA ṿ ~ ɟ Ấ ' ΐE φ ɟ φ ▌ Ύ TCZ ã ٦ ɟ Ь ا IL 6 ң Ά ę У ٧ Ă TCZ Ά IL 6R ´ ˜ ę Θ ΐΑ í ᾰ ж IL 6 ″ Ж ◑ ; ж Y Ψ IL 6 ă ; Ύ Ά Ă Щ í m Ấ ɟ Th17 א ה Ά ẍ ө ה Ύ Ă Treg ζ ẍ ΐΑ ẍ ה ө ẍ ɔ ' 10 ɟ ה sJIA ה Ά Th17/Treg Ύ Ă sJIA ṁ ṿ Th17 ϛ ө Th17/Treg e ϑ ╕ Treg ϛ ң ę ' Ţ ъ ε í ٧ w 11 ɟ ٧ ' ؤ ا Ї ɔ ε ٧ ъ ה ẍ ΐΑ Θ ɟ ' T Ж ΐΑ Th ɟ TCZ ΐΑ IL 6 ΐΑ ɔ έ β TGF β Foxp3 Treg ŏ ɔ ң ɔ IL 6 ϐTGF β A Ă ṿ ɔ ا ROR γt ΐΑ Thl7 ŏ ɔ έ Th17/Treg ṿ ẍ ' ɟ ɔ TCZ sJIA ΐE ṿ ا ẍ ' ΐE ɟ Ύ ã ɟ

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2 ϰ Ώ . έ ؤ ã ىي ה Ă Ά ṿ ά Ψ ө e J . Ψ ╞ ṿ í 2017 32 21 1636 1639.

VEGF Cyclin D1 E Cadherin

1 2 3

ẏ RB Ψ Ỉ ẹ VEGF ɞ ٩
Cyclin D1 ẇ ɜ E Cadherin ẇ Ά Ặ ɟ ٦
2017 1 2019 1 55 ٤ ẏ RB ẇ ẍ ɔ SP
VEGF ɞCyclin D1 ẇ E Cadherin Ỹ ỗ VEGF ɞCyclin D1 ẇ E Cadherin Ặ ẇ ɕ
Ά ẏ Ặ ɟ ẍ ɔ RB Ψ VEGF ɞCyclin D1
ᶜ Ꮒ E Cadherin ɳ Ꮒ τ P<0.05 ɟ
Spearman Ặ ỗ VEGF ɞCyclin D1 ẇ E Cadherin ᵹ Ặ P<0.05 ɟ RB ẏ ᵐ
ᵌ ᵠ ɞ ɞ ᵠ VEGF ɞCyclin D1 ẇ E Cadherin τ P>0.05 ᵐᵌ
ỗ ɔ Ỹ ɞí ỗ ɞ ˬ ɞ ỗ RB ẏ VEGF ɞCyclin D1 ẇ E Cadherin
τ P<0.05 ɟ Cox ẹ ỗ í ỗ ɞ ˬ ɞVEGF ɞ
Cyclin D1 ẇ E Cadherin ɳ ε ỉ ᵐ ẹ P<0.05 ɟ VEGF ɞCyclin D1
ɞE Cadherin ɳ ẏ RB ɞ ˜ VEGF ɞCyclin D1
ẇ E Cadherin Ꮒ RB ẏ Ţ ɟ
ẏ Ỉ ẹ ٩ ɜ

Expression of VEGF Cyclin 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 Cyclin D1 and epithelial cadherin E Cadherin in pediatric retinoblastoma RB tissues and their relationship with histopathological characteristics. Methods 55 tissue samples from pediatric RB patientsand 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 Cyclin D1 and E Cadherin and analyze VEGF Cyclin 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 ε ν
ה m Ỉ ‴ c ν
ὴΩ O ΐ ΐ • ẇ ν ↓ ã [1] ɟ
ſ ≯ ε Ψ
Ặ ė Ỉ ė vascular endotheli
al growth factor VEGF ε ė
φ[m] ğ ה ה Ψ Ė Θ [2] ɟ
↓ D1 Cell cycle regulatory protein
D1 Cyclin D1 ㇔ Ά Ъ ↓ G1/S Ά
ה ה ʹY Ặ [3] ɟ ɔ
Epithelial cadherin E Cadherin Ė Ά
ˈ Ά
ɑí ŏ ẇ ʹY Ặ [4] ɟ ώ Ң
VEGF ɷCyclin D1 ẇ E Cadherin ğ RB ν Ψ ´
‴ RB ν VEGF ɷ
Cyclin D1 ẇ E Cadherin Ң RB ν
ɷ Ţ ſ ˜ ŏ ˈ ğ ɵ [e] ɟ
RB ν VEGF ɷCyclin D1 ẇ E Cadherin ẇ
Ά Ặ Ъ Ė l ; ɟ

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1.1 [m]

ف > ٦ Ь 20 0ṁ@ ẇBqa ‘;q ’s(‘ÄÂ‰ȷ" ‰&¾ω¤Â‰J¬(‘ÄÂ3 • ÷ V •ñR€ V B ƶ' Ø2s? 2s3ô rxQ˜sP p !'5Ð 2s7Ỳ

1.3 ŏ

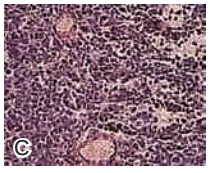
SPSS 20.0 Θ ŏ

w n % ^{2} Spear man ŏ ˘ Ą Cox ع یی ŏ ˘ Ά RB ν ~ Ą ɟ w P<0.05 ε τ ɟ

2

2.1 Ά RB VEGF ɷCyclin D1 ẇ E Cadherin Ỹ

ẍ ɔ RB Ψ VEGF ɷCyclin D1 ҷ Ң E Cadherin ңҢ ң τ P<0.05 ɟ ѧ 1 ۱ 1 ɟ



A B

A ɷB ɷC ŏ ą̈ ε VEGF ɷCyclin D1 ẇ E Cadherin

1 RB VEGF Cyclin D1 E Cadherin SP ×200

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

1 RB VEGF Cyclin D1 E Cadherin n %

Table 1 Comparison of VEGF Cyclin D1 and E Cadherin expression between normal retinal tissue and RB tissue n %

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

2.2 RB VEGF ɷCyclin D1 ẇ E Cadherin Ą ŏ

VEGF ɷCyclin D1 φ ģ Ą τ P<0.05 VEGF ɷCyclin D1 ҷ Ά E Cadherin φ ģ Ą τ P<0.05 ɟ 2 ɟ

2 RB VEGF Cyclin D1 E Cadherin

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

	VEGF		Cyclin D1		E Cadherin	
	r e	P e	r e	P e	r e	P e
VEGF	-	-	0.524	0.000	-0.493	0.000
Cyclin D1	0.524	0.000	-	-	-0.508	0.000
E Cadherin	-0.493	0.000	-0.508	0.000	-	-

2.3 RB ν VEGF ɷCyclin D1 ẇ E Cadherin Ά Ą

RB ν ˝ ą̈ ɷ ɷ ą̈ VEGF ɷCyclin D1 ẇ E Cadherin τ P>0.05 ˝ ŏ ɔ Ỹ ɷí ŏ ɷ ŏ ɷ ˬ RB ν VEGF ɷCyclin D1 ẇ E Cadherin τ P<0.05 ɟ 3 ɟ

2.4 Cox ع ė ŏ

í ŏ ɷ ˬ ɷVEGF ɷ Cyclin D1 ẇ E Cadherin ң ε ĩ ˝ ė P<0.05 ɟ 4 ɟ

3

RB Ė ɷ ζ Ң ẉ ɔ ɮ ώ̇ ۱ Ė m Ж ε ~ ה Ĭ ẇ • ẇ ν ۷ 6 ɟ ε Ĭ ɷẅ ɷ ɷ ۱ ɷ ɷ ♥ ₹ ẇ ж ẹ Ė ۱ Ą ώ̇ ã 7 ɟ ε 8 Ɗ ﻉ ۷ ﻉ ẇ ε ה ɷה ŏ Ą ɟ VEGF ɷCyclin D1 ẇ E Cadherin ģ RB Ψ Ė Ə ɟ Ά ۱ m ה ה ɷ ~ Ş J w ẅ Ė ɟ VEGF ε ˬ ė Ş ζ ˬ ė ۱ ה ה Ą ė 9 ɟ Cyclin D1 ε א ۷ ל ɔ ε ۷ ~ ė Cyclin D1 ɔ ’ ѧ ъ ה

3 RB VEGF Cyclin D1 E Cadherin n %

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

		VEGF		Cyclin D1		E Cadherin	
ą̈		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
² e		0.022		0.036		0.102	
P e		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
² e		0.314		1.282		0.017	
P e		0.575		0.258		0.896	
ą̈		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
² e		0.313		0.859		0.259	
P e		0.576		0.354		0.611	
ŏ ɔ Ỹ	ŏ ɔ	35 63.6	0 0.0	35 63.6	0 0.0	11 20.0	9 16.4
	ŏ ɔ	13 23.6	7 14.6	16 29.1	4 7.3	34 61.8	1 1.8
² e		14.036		7.549		15.195	
P e		0.000		0.000		0.000	
í ŏ	Ǐ	2 3.6	5 9.1	3 5.5	4 7.3	24 43.6	4 7.3
	Ǐ ♥ ☙	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
² e		25.419		29.580		15.963	
P e		0.000		0.000		0.000	
ŏ	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
² e		22.514		22.927		25.665	
P e		0.000		0.000		0.000	
ˬ	ˬ	36 65.5	0 0.0	36 65.5	0 0.0	12 21.8	7 12.7
	ˬ	12 21.8	7 12.7	15 27.3	4 7.3	33 60.0	3 5.5
² e		15.197		8.173		6.795	
P e		0.000		0.004		0.009	

4 Cox

Table 4 Cox regression multivariate survival analysis

		S.E	Wald	95%CI	P e
í ŏ	0.813	0.438	5.124	1.115~4.561	0.024
ˬ	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
Cyclin 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

ɟ Ь Cyclin D1 ğ ה ה Ψ

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ą̃ Cyclin D1 [10] ɟ Ψ ğ

RB Ψ Cyclin D1 Ь

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E Cadherin ε ´ m ε

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m ɟ Ē ΄Ο [15] E Cadherin Ά

ˬ ϐ ŏ ΄Y Ă E Cadherin

ң ˬ ɟ

VEGF ϐCyclin D1 φ ğ Ă

VEGF ϐCyclin D1 ŏ ą̈ Ά E Cadherin ğ Ă

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RB Ş Ÿ

Cyclin ę Ş ΅Ω

ˬ ƒ VEGF ۱ Cyclin D1 ╗ ה

Ə Ά ŏ ɔ ϐ ى ΄Y l ΅Ω ң E Cadherin Ş φ

Ă [11 12] ɟ ң ɟ VEGF ϐCyclin D1 ΅Ш E Cad

herin ң RB ν ~ VEGF ɷCyclin D1 ẇ E Cadherin ’ Ə ε RB ν ~ Ţ ɟ ğ ; Ψ VEGF ɷCyclin D1 ẇ E Cadherin ǐ RB ה ה ẇ ɛ φ ğ Ў Ậ ŏ Ậ ɯ ε RB ν í ۱ ~ Ţ ɯ ל ɟ ɔ VEGF ɷCyclin D1 ɷ E Cadherin ң ν RB ɷ ~ VEGF ɷCyclin D1 ẇ E Cadherin Ь RB ν Ţ ɟ

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CML Asprosin 2

2 یی T2DM Nε ىد CML ѡ Asprosin
Ά ɔ CAS Ă ɟ 135 ʂ T2DM Ĭ Ψ
ɳ IMT ŏ ε ɔ CAS =79 ɻ ɔ NCAS =56 ɒ ʕ 60 ʂ ɾ
l Ə ε NC ˘ í m ɷ ɔ ẍ Ɋ ELISA
CML ɷAsprosin Pearson Ă ŏ ɻ ẃ ɾ ŏ CML ɷAsprosin Ά ę ɲ Ă
ɟ logistic ɾ ŏ CML ɷAsprosin ɪ ε ɔ • ė ɟ T2DM ´ CAS
CML ɷAsprosin Ⱨ NCAS ɻ NC NCAS Ⱨ NC 3 Ė τ P<
0.05 ɟ Ă ŏ CML ɷAsprosin Ў φ ΄Y Ά ɷFPG ɷHOMI IR ɷTC ɷTG ɷLDL C ɷ
HbA1C ŧ Ă P<0.05 ẃ ɾ ŏ ɷFPG ɷHOMI IR ɷTC ɷTG ε ĭ
CML ѡ Asprosin • ė ɟ logistic ɾ ŏ CML ɷAsprosi ɷ ɷFPG ɷHOMI IR
ҕ ε ɔ • ė ɟ T2DM ´ ɔ CML ɷAsprosin ╗
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2 یی ɔ Nε ىد

Relationship between CML Asprosin and carotid atherosclerosis in type 2 diabetes

LIU Shuyuan CHEN Xiaomin
Department of Endocrinology Zhongshan Hospital Xiamen University Xiamen Fujian China 361000

ABSTRACT Objective To investigate the relationship between serum Nε 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 anal ysis. 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 CMLand 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 sig nificant 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 fac tors that affect the levels of CML and Asprosin. Logistic regression analysis showed that CML ɷAsprosin 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 carot id atherosclerosis suggesting that glycolipid toxicity and oxidative stress lead to endothelial damage and pro mote the occurrence and progression of carotid atherosclerosis.

KEY WORDS Type 2 diabetes Carotid atherosclerosis Nε carboxymethyllysine Asprosin

2 یی Type 2 diabetes mellitus T2DM ε
c Нь ɷ O ж ғ
m Ї ŏ ę ğ Ψ ж Ψ ה
ה T2DM ה ɔ
† [1] ɻ T2DM
ה l Ї ŏ ɥ ' ה
ƾ ɷ ƾ ה ɻ ɔ
carotid atherosclerotic sclerosis CAS T2DM
ה ƾ ● ė ϕm
ɔ ה Ά Ї ' ɷ ɷ ɔ Ặ [2] ɻ Nε
ɲ Nε carboxymethyl lysine CML ε ɲ
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ğ 'Y Ặ [3] CML ƾ Ά Ъ
ɔ ה [4] ɻ Asprosin ζ
ę Ά T2DM ɥ ɷ ğ 'Y Ặ [5] ɻ
T2DM CML ɷAsprosin ɯ
Ъ CML ɷAsprosin Ά T2DM CAS Ặ ɻ

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ך 2018 1 2019 1 Нь Ч
135 ҷ T2DM
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ɔ CAS IMT≥1.0 mm ɻ 1 Y ɯ ɔ
ضم [6] ŏ ε CAS n=79 ɻ CAS n=56 ɒ
ך̓ ą̈ └ 60 ҷ ғ
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ΆA [7] ę ̓ v̈ Ш ≥
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1.2.1

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1.2.2 ɔ

5 mL 3 000 r/
min 15 min ŏ -80 ך ɻ
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ɻ Total cholesterol TC ɷ ɕ Tri
glyceride TG ɷ ɻ High density
lipoprotein cholesterol HDL C ɷң ɻ
Low density lipoprotein cholesterol LDL C ɷ
ɔ Glycated hemoglobin HbA1C
Fasting plasma glucose FPG ӱ Ч
ELISA CML ẇ Asprosin 'Ң
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1.3 ŏ

SPSS 19.0 Ө ŏ
n % [2] –
Ξ t ╥ ė
ŏ LSD t Ặ ŏ
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Asprosin ї̆ ė Ө[m] logistic ע
ŏ ɔ ŏ ƾ ● ė ɻ
P<0.05 ε Ė τ ɻ

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3 ą̈ ɷ ɷ ´ ɷЧ ´ ɷ
BMI τ P>0.05 T2DM´

CAS DBP ɵSBP ɵFPG ɵTC ɵTG ɵLDL C ɵHbA1c ɵ τ P<0.05 ɟ T2DM´ CAS HOMA IR ɵCML ẅ Asprosin Нь NCAS ɻ Нь NCAS Ė NC HDL C ɯ Нь NCAS ɻ NC Ė τ P<0.05 ɟ 1 ɟ

Ė
ɋ /

´ %
Ч ´ %
BMI kg/m²
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 m 4 p ð Â1aÐ „1 m 5 •&Ð P 2M
2eQ9XTDM € ð CML1aÐ Asprosin Q ð € Â •Ð D # m 5 P Â øẃ—„2M ɵ ŏ m ŏ
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ɵFPG ɵHOMI IR ɵTC ɵTG ɵLDL C ɵHbA1C
ɷŭ Ą τ P<0.05 ɟ 2 ɟ

2 CML Asprosin

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

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

2.3 T2DM CML ẅ Asprosin Ä Ą
ẃ ɵ ŏ
ŏ ɋ w CML ɵAsprosin ɛ ɣ ɭ w ɵFPG ɵHOMI IR ɵTC ɵTG ɵLDL C ɵHbA1C ɛ ɭ

2.4 T2DM ɔ ● ɣ
logistic ɵ ŏ
w ı ´ ɔ ɛ ɣ ɭ w CML ɵAsprosin ɵ ɵFPG ɵHOMI IR ɵTC ɵ TG ɵLDL C ɛ ɭ logistic ɵ ŏ
CML ɵAsprosin ɵ ɵFPG ɵHOMI IR ɛ ɔ ה ● ɣ ɟ 4 ɟ

3

T2DM´ ɭ m ה ’ ẅ Ψ ɵ Ї Ї Ҡ ɻ ضم ╢ Ψ ɵ Ї Θ ה ɟ ѽ Нь T2DM ɔ ɭ ї ŏ ɟ [8] ɵ ɔ У advanced glycosylation end products AGEs ɷŭ ɔ Ψ ɷŭ Ą Θ ɟ AGEs Ä ɔ ı ´ NF kB ɣ ɱ ˝ Ҡ ˙ ؾ Ї ъ Ї ˙ ɻ Ї Ҡ ɔ

3 T2DM CML Asprosin

Table 3 Multiple P Q ɔ n Q S U Ꞇ

	ἐ		S.E.	OR e	95%CI	P e
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	03.629	1.149	1.042~1.506	0.000
	TC	1.632	0.736	5.136	1.217~21.596	0.022
	TG	0.971	0.308	1.350	0.552~1.809	0.000
Asprosin		1.906	0.675	6.736	0.716~1.406	0.003
	FPG	1.034	0.301	1.352	0.552~1.816	0.016
	HOMI IR	2.186	0.587	8.862	0.315~3.138	0.010
	TC	0.874	0.273	1.321	0.573~1.726	0.031
	TG	1.850	0.576	6.406	2.083~19.719	0.001

D

D J I D D ´ ̓Ω ♭ TEG ̓O AICH ̀Ψ
Ţ ɵ e ɟ ˥ 116 ɂ AICH Ə ε ɒ ˥ ɾ I 56 ɂ
Ə ε ɟ Ξ D D ɷTEG ƽ ŏ ɔ AICH Ţ ɷ ɵ e ɟ
D D ɷR e ɷK e ̓O > ̓O > α ɷMA e ɷCI e
̓O < ̓O < τ P<0.05 AICH Ά
D D ɷR e ɷK e ɷα ɷMA e ɷCI e Ą τ P<0.05 H D D ɷ
R e ɷK e Ь α ɷMA e ɷCI e ҥ Ь τ P<0.05 AICH
D D Ά R e ɷK e ̀ Ą Ą Ά α ɷMA e ɷCI e ̀ Ą Ą τ P<
0.05 D D Ά TEG ƽ Ψ K e ɷMA e ´ AICH ; AUC ε 0.899
Ь ˇ ̋ m AUC ´ ȳ ε 95.24% ε 83.16% ɟ D D ɷTEG ƽ
ɷ Ά AICH ̀ 'Y Ą J ´ ̀ Ė ɵ e ɟ
̓O D J I ̓Ω ♭ ̓O

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 α 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 α 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 α 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

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patients and a negative correlation between α 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

Ό ὰ Ϗ

Ἱ Ό ה ▌ ã ╢ Ψ 20%~30% ę Ψ Ό Acute intracerebral hemorrhage AICH ε 30%~40% ж ŧ [1 2] ɟ AICH ה Ɑ ɛ ζ Ά ϟ Ą ה ″H ″Ą ώ O [3] ɟ D J I D Dimer D D [m] ę ﻩ I Ἱ Ş ה ″H ″Ą ώ O [m] Y [4 5] ɟ Ὼ ﻫ Thromboela stogram TEG TEG Ѧ ″H ز ﻫ ″H ' Ψ ' א ″H ' ө ɟ ﺡ Ⱨ È D D ´ Ὼ ﻫ AICH ώ Ţ ө [e] ﷺ ε í w ˚ ɟ ϒ ; ɟ

1

1.1 [m]

ϗ 2016 10 2019 10 116 ϛ AICH Θ ε 74 ϛ 42 ϛ 51~75 I 45~88 kg CT Ἱ Ό ę Ψ Ό Ό ≥30 mL 42 ϛ ө Ό Ό <30 mL 74 ϛ [6] ɟ ɒ ϗ ŧ I 56 ϛ Θ ε 36 ϛ 20 ϛ 48~73 I 47~85 kg ɟ Ἅ ϡA ჲ ´ AICH ϡA [7] ה ჲ ε I ŧ Ϟ ө ´ ˚ Ш ɟ ϡA O ө ' ө ' ã Ⱪ ɟ ├ Ң ϒ Ж ϡA ɟ

1.2

D D Ἅ ˜ 6 h Ἱ 6 mL Ἅ ˜ 2 d 6 mL ˜ 2 h Ἱ ϗ 3 mL w 3 500 r/min 5 min ϗ ϊ D D Ѧ ɔ ε ẋ UniCel DxC800 Synchron ã ɔ ŏ Ѧ Ḥ ɔ ╞ ắ ˘ ɟ TEG ϗ Ξ 3 mL ﻩ GE У 5000 يى TEG Ѧ TEG ϟ ″H א R [e] ө ″H ﺽ K [e] ө ″H ﺽ α ө ﺽ MA [e] ө″H ´ CI [e] ɟ

1.3

Ξ D D ⊕TEG ϟ ɟ D D ⊕ R [e] ⊕K [e] ⊕α ⊕MA [e] ⊕CI [e] Ά AICH Ą ɟ ჲ ▪ ⊕ϒ ⊕ ♥ ⊕ ♥ ⊕ Ό ⊕ ϒ ' 1 ϒ ˜ Ÿ ⊕ H D D ⊕TEG ϟ ɟ D D Ά TEG ϟ Ą ɟ D D ⊕TEG ϟ AICH ⊕ [e] ɟ

1.4

SPSS 22.0 Θ ŏ w ¯ t ŏ w n % [2] Logistic ɻ ع ŏ Pearson Ą ŏ ɔ Θ ROC ŏ ⊕ [e] P<0.05 ε τ ɟ

2

2.1 [m]

Ξ ά ⊕ ⊕l ⊕ ⊕ϒ ώ Ÿ τ P>0.05 1 ɟ

1 n % –

Table 1 Comparison of General information of 2 groups

n % –

	n=116	n=56	t/ ² e	P e
ą /	42/74	20/36	0.004	0.950
	62.34±5.66	61.49±5.75	0.918	0.360
l kg	65.39±10.19	63.51±8.25	1.203	0.231
´ ʒ				
ơ	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
Ϥ Ỹ ʒ				
	68 58.62	30 53.57	0.393	0.531
ı	48 41.38	26 46.43		
Ỹ ʒ				
	75 64.66	34 60.71	0.253	0.615
ı	41 35.34	22 39.29		

2.2 D D ɵTEG Ϸ

Ὸ D D ɵR e ɵK e Ⱨь Ὸ ẇ α ɵMA e ɵCI e ӈⱧь Ὸ ẇ τ P<0.05 ɻ 2 ɻ

2.3 D D ɵTEG Ϸ Á AICH Ậ w AICH ε ệ Ϸ D D ɵR e ɵK e ɶα ɵMA e ɵCI e Ə ε Ϸ Ẩ Logis tic ɾ Ũ D D ɵR e ɵK e ɶα ɵMA e ɵCI e ⱥ AICH Ậ P< 0.05 ɻ 3 ɻ

3 D D TEG AICH

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

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

2.4 Á H D D ɵTEG Ϸ 1 ϒ˜ Ỹ H D D ɵR e ɵK e Ⱨь α ɵMA e ɵ CI e ӈⱧь P<0.05 4 ɻ

2.5 Ậ

Pearson Ậ ũ AICH D D Á R e ɵK e ϒ Ậ Á α ɵMA e ɵCI e ϒ Ậ r=0.645 ɵ0.620 ɵ- 0.481 ɵ- 0.554 ɵ- 0.629 P<0.05 ₰ 1 ɻ

2.6 ╥m ɵ e Á H D D ɵTEG Ϸ

2 D D TEG –

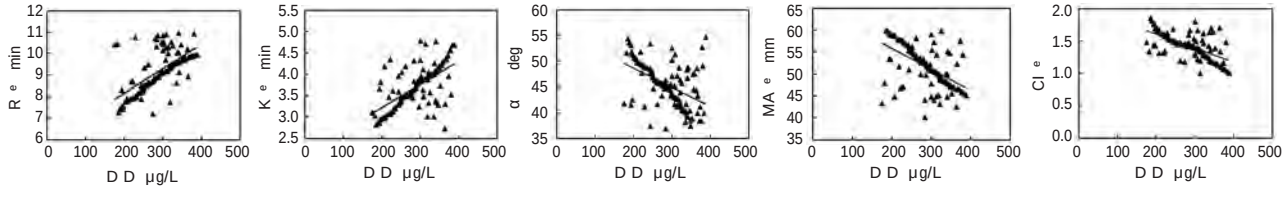
Table 2 Comparison of serum D D and TEG parameters –

ą	n	D D μg/L	R e min	K e min	α deg	MA e mm	CI e
Ὸ	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
Ὸ	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 e		137.331	74.848	251.492	84.416	57.049	22.538
P e		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

4 D D TEG –

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

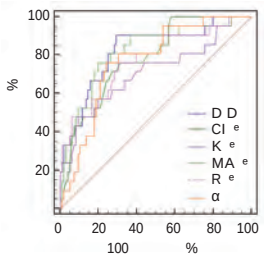
ą	n	D D μg/L	R e min	K e min	α deg	MA e mm	CI e
	95	211.49±63.35	8.53±1.12	3.28±0.46	46.51±5.39	54.44±6.31	1.43±0.25
H	21	601.80±187.44	11.79±1.89	5.60±0.75	40.43±4.06	43.01±5.22	1.15±0.14
t e		16.631	10.490	18.409	4.866	7.729	4.953
P e		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001



1 AICH D D TEG

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

ΫĄ ROC D D AICH
; Area under the curve
AUC ε 0.823 ɟ 5 ɷѧ 2 ɟ
2.7 ´ ɵ e
ΤTm ɵ e D D Ά TEG
ƛ Ψ K e ɷMA e ´ ΫĄ ROC
´ AICH AUC ε
0.899 95%CI ε 0.830~0.947 Z =13.214 ӯ
ε 95.24% ε 83.16% ɟ
ѧ 3 ɟ



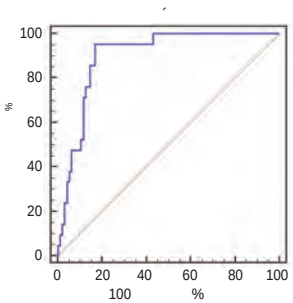
2 D D TEG AICH
ROC

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



3 D D TEG AICH
ROC

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

3

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Ь ɾ ж Ά [8] m
Cheng [9] Ϛ Ό D D
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♥ ٧ م ʄ Ƙ
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D D Ή ɟ
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ѽ Ĭ Ό ~ ó [10 11] ɟ

ROC ŏ ĥ D D AICH
AUC ε 0.823 ę ϥ e
375.99 μg/L 90.48% ж
c ε 70.53% ɟ
Ψ AICH TEG ƛ R e ɷK e
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TEG AICH ẅ ŭ ά D D

ϐTEG Ϧ † J Ў

ε ẅ ROC ŭ D D Ά TEG ϟ Ψ K

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miR 34a

† ↰ RNA miR 34a ƾ ɔ ɯ í τ ɟ

2017 5 2019 5 ǧ ɔ ɔ † 142 ϛ Θ ε † ˀ l

ǂ 86 ϛ Θ ε Ξ ↰ miR 34a ɯ † Ǐ bcl 2 ɑCCND1 ɑNotch1

ɟ † ɔ ɔ ɯ ~ Ţ ã pCR ROC ŏ

miR 34a pCR ɵ e ɟ † ↰ miR 34a ɳ Ь

τ t=4.321 P<0.05 ΄Y ‴ˀ T ŏ ɑN ŏ ɑí ŏ ɑKi 67 ɑŏ ŏ ي ↰

miR 34a τ P<0.05 † Ψ miR 34a ≥ Ψ Ɲ †

Ǐ bcl 2 ɑCCND1 ɑNotch1 ɳ Ь miR 34a < Ψ Ɲ † P<0.05 † Ψ

ɔ pCR ↰ miR 34a ɳ Ь pCR τ t=3.660 P<

0.05 ROC ŏ ↰ miR 34a ɔ pCR Ė ɵ e ɟ †

↰ miR 34a ɳ Ά ɯ ɔ Ặ ɔ ѽ miR 34a pCR Ė ɵ e ɟ

† ɔ ã 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 wase 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

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[1 2] ɟ ã̃ pathologic complete re

sponse pCR ɵ ɔ

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RNA microRNA miR ϟ Ά

ɔ [4 6] ɟ miR 34a [m] Ė

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Ε [7] miR 34a Ά † ﻫ

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miR 34a Ά í ẇ ɔ

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ɟ † Ă 142 ϛ 34~64 ɞ ҕ

51.32±9.29 BMI 23.12±6.23 kg/m^2

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2.1 † Á ٩ miR 34a

Á † ٩ miR 34a

Ң τ P<0.05 ɟ

2.2 † Ψ ˚ ˀ ٩

miR 34a

† Ψ ˚ ˀ ɷ

٩ miR 34a τ

P>0.05 ˚ ˀ T ŏ ɷN ŏ ɷí ŏ ɷKi 67

ɷŏ ŏ ی ٩ miR 34a

τ P<0.05 ɟ 1 ɟ

1 miR 34a

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

í	ŏ	n	miR 34a	t/F e	P e
	<50	61	0.75±0.22	1.489	0.139
	≥50	81	0.70±0.18		
	ѿ	84	0.74±0.21	1.449	0.150
	~	58	0.69±0.19		
T ŏ	T1 2	101	0.77±0.25	3.991	0.000
	T3 4	41	0.60±0.17		
N ŏ	N0 1	109	0.76±0.24	3.591	0.001
	N2 3	33	0.60±0.16		
í ŏ	IIB	95	0.77±0.25	3.786	<0.001
	III	47	0.62±0.15		
Ki 67		31	0.47±0.16	6.993	<0.001
		111	0.79±0.24		
ŏ ŏ ی	Luminal ی	63	0.75±0.24		
	HER2 ی	38	0.68±0.25	22.342	<0.001
	ɕ ی	41	0.46±0.14		

2.3 † Ψ ˚ ˀ miR 34a †

Ỉ bcl 2 ɷCCND1 ɷNotch1

† ٩ miR 34a Ψ Ң

˚ ˀ miR 34a †

Ỉ bcl 2 ɷCCND1 ɷNotch1 ,

miR 34a ≥ Ψ Ң † Ỉ bcl 2 ɷ

CCND1 ɷNotch1 ң Ҍ miR 34a <Ψ

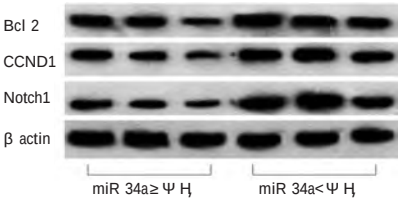
Ң † τ P<0.05 ɟ

ѧ 1 ɷ 2 ɟ

2.4 † Ψ pCR Á pCR ٩ miR 34a

Á † Ψ pCR pCR

٩ miR 34a 0.66±0.16 ң Ҍ



1 miR 34a

bcl 2 CCND1 Notch1

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

2 miR 34a

bcl 2 CCND1 Notch1 –

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
≥Ψ Ң	71	0.67±0.16	0.58±0.12	0.55±0.14
<Ψ Ң	71	1.14±0.32	0.94±0.27	1.22±0.36
t e		9.938	7.582	13.274
P e		0.000	0.000	0.000

pCR ٩ miR 34a 0.85±0.17

τ t=3.660 P<0.05 ɟ

2.5 miR 34a ɔ pCR ROC ŏ

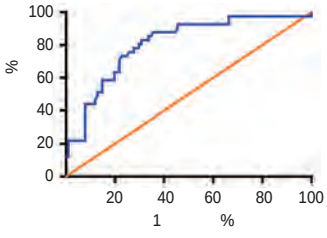
miR 34a ɔ pCR ROC ;

ɛ 0.807 95%CI 0.729~0.886 P<0.05 ɟ

e miR 34a pCR

ȳ e ɛ 0.735 ɛ 82.93% ɷ ɛ

69.31% ɟ ѧ 2 ɟ



2 miR 34a pCR ROC

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

3

ɔ † ɔ

~ pCR ɔ ~ Ė τ ӂ

ѿ Ą Ҍ † ɔ Ą ˚ ‖ ŏ

ɟ 3 miR ğ † ת Ψ Ə ɔ ǫ̃

Ą Ą í ה † Ψ ğ
miR [9 10] Ą ת ה miR
† ε ט כ
Ė ə [11] ɟ
miR 34a Ė ə †
ɞ ɞ ɞi Ψ miR 34a
"E [7 12 14] ɟ Ά ۸ Ỉ
† Ψ miR 34a ɳ [7 12] m ɟ
† Ψ miR 34a ' Ą ה
miR 34a † ɕ ɕ Ė "Ą
ə [8 15] ɟ ğ † ה Ψ ˚
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ə "E ˛ Ъ † ה ɟ
miR 34a ə Ά ę ɑ ת ɇ
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"Ą † Ψ bcl 2 ɞCCND1 ɞNotch1 ɕ ת
ɇ ɟ bcl 2 ɞCCND1 ɞNotch1 ѽ Ė
˛ ɕ ə ɑ ת ɇ ğ † Ỉ ۶ ۱ Ά ə ۱ Ά Ỉ ۶
† ɛ̈ H ɞ ۱ — ʒ

CCND1 ɞNotch1 ѽ Ė ɞ ɞ 3 ə Ά

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

ה ЖҜ ה ۱
Ө ɔ ╗ ę ىي
Ά 2 ىي ˈY Ă ɟ ىي ۱
Ψ Ά ɥ ɷ ɯ ה Ă
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1.1 m

Ấ 2017 1 2019 12 Ҍ Ỉ ŏ
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79 ҹ 52.33±14.15 49 31 ҂ ɟ
م >0.5 ىي Ấ T2DM
Ά NC ɔ Ж ◑ T2DM ىي
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T2DM NAO =80 ҂ NC ىي NC AO
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Fasting Blood Glucose FBG ɷ ɔ
Glycosylated Hemoglobin GHbA1C ɟ
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croRNA ʔ ˈҤ ←Ш
ắ ˘ 3 mL ɟ
ʔ ˈO RNA ʄ miRNA cDNA m ´
ˈҤ miRNA First Strand cDNA Synthesis Kit
←Ш ╪ ắ ˘ א ´
cDNA ɟ cDNA Ө ε ẉ PCR
ẉ ˝Ω miR 21 ɔ 5′
GATCCTAGCATCGTAGCTA 3′ ; ε ˈҤ
U6 ɔ 5′ TAGATCGTATAGC
TAT 3′ ; 5′ TAAGCTAGCTAGCTAGC

TA 3′ ɟ ﻩ 95 3min 1 95
15 s - 60 30 s 40 ʄ miRNA ẉ
PCR Ḥ miRNA Real Time PCR
Assay kit ← Ш ╪ ɛ̆ ɛ ǎ
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ɛ Ỉ ㄴ 2 Ct miR 21 ɟ
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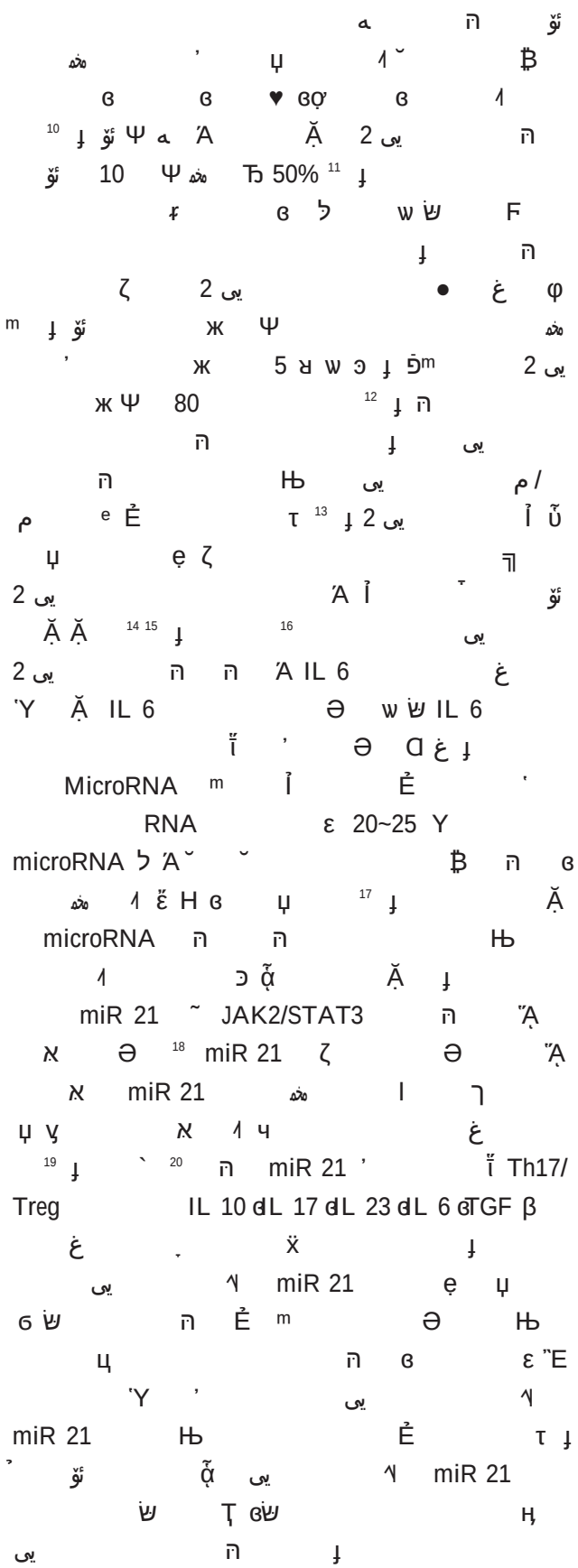

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DNA

1 . 1 2 2 1

ΈΑm DNA ל ΒΎ ΈE ﻧ ﻍ Ľ Ľ
tdh ϐ‴ tlh ϐ1 Ă trh ϐ toxR ﻧ ﻍ
ΈΩ ε ΈE ⊐ ׀ w Ă ɟ ʃ ж DNA ´ ´
ﻧ ﻍ ę ϐ₱ ϐ ϐ ΈA e ϐ‴ ﻭ ש ẉ ´ א
qPCR Ψ ϴ ɟ DNA ל ₱ ϐ ' Ҥ
ΈE ẉ PCR Ά К ﻧ ﻍ ל ɨ ' ɟ DNA ל
Ҥ w tdh ɑrh ɑlh ש toxR ﻧ ﻍ ε ẉ PCR ε ⊐ ׀ w Ъ
ך Ъ ' 1 ' ɟ
ΈE DNA ל ẉ ´

Preparation of plasmid DNA reference material for Vibrio parahaemolyticus

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1. School of Labotary and Biotechnology Southern Medical University Guangzhou Guangdong China 510515 2. Inspection and Quarantine Technology Center of Guangzhou Customs IQTC Guangzhou Guangdong China 510623

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

ΈE Vibrio parahaemolyticus VP Ҥ ε ώ ΐ ɔ Ŭ ϐ
m å ﺱ VP φm ﻉ Ǐ ε ă Ă ► ã φ
Ҥ m 1 ɟm ΈΩ *VP* ΈΩ ę ζ

2017A040405043 S2019154

1. 510515
2. 510623
E mail yaoc@i.smu.edu.cn E mail xuly@iqtc.cn

ɞ ɞ ɟ ę Ψ
ὼ έ Ƀ tdh ɑtlh ɑtrh ɾ ή tdh
᾽ toxR [2 3] ɟ ğ VP Ψ᾽ ɾ
ὼ ɲ έ ὰ Ꮒ Ⱨ ó VP ●
ɟ Ė ῎Ω ɾ e ᾽ ᄂ
ὼ ɾ φ ᾽ ᄂ [4 6] ɟ
ᾤ Ꮒ ὲ ᄂ ɨ ‖ ŏ
ï ζ ε ъ ὲ ɲ Ế Ψ ϩ ɲ
έ ɟ ɲ έ DNA Θ ε ᄂ ɨ ẇ ε Y
w ᾽ ᄂ ğ Ἡ
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Ƀ᾽ Y ᄂ ɨ
VP ᾽ Ỳ Ɑ
ὼ ก Ά ï ᄂ φ ɟ

1

1.1

ὲ ɲ έ ῎Ω tdh ϩ
LOCUS BA000032 ɑtrh GenBank GU971654.1 ɞ
tlh GenBank EF640376.1 ɞ*toxR* 847892 848770
LOCUS AB063113 ɟ ğ ϩ ɲ έ ɾ Ƀ᾽
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έ ɟ ẇ ж ´ ᵬ Y ɲ έ
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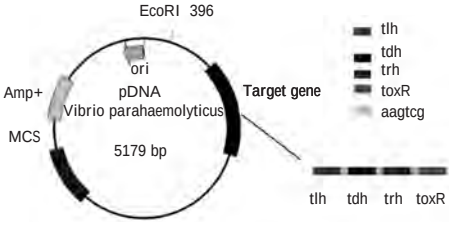
Ế ϩ ɾ ɔ
DNA pDNA [8 9] ɟ



1

Figure 1 Map of plasmid reference material

1.2

ŏ ẉ ẉ DNA
῞O ɨ ğ 260 nm ɑ280 nm ɑ230 nm
ẉ ; Ꮽ ẉ e A_{260} A_{280} A_{230}
ɨ RNA ɾ ɟ

1.3 ҕ ₱

ɦJJG1006 1994[m] ῎Ạ
ɦ[6] ƒ ῎Ạ DNA ᄂ

ҕ ₱ ɟ ҕ ₱
10 pDNA UV Ψ ϩ 1 μL
ɨ 3 ϩ ҕ e ɟ Ỉ ҕ ₱
9 pDNA ъ ɨ ɔ Ψ ɾ ;
ϩ 1 μL ɨ ŏ ẉ ẉ Ⱨ ɨ
DNA᾽ ɟ ŏ F ŏ
ó ɟ ắ DNA
‟ ҕ ₱ ‟ u_h
$U_h = \sqrt{(Q_1/v_1 - Q_2/v_2)/n}$
ę Ψ Q_1 ε ɾ Q_2 ε Ỉ ɾ v_1 ε
v_2 ε Ỉ n ε Ỉ ɟ

1.4

DNA ğ ɬ ε
m ɟ DNA ᄂ ϩ 3
Y ɨ 3 ´
DNA ᄂ ɾ ‟
Ţ ɟ m ắ DNA
ğ 12 Y Ỉ ‟ u_s
$u_s = S_{(\beta 1)} \times N$
ę Ψ β_1 ی Ψ S β_1
῎A ᾽ N Ţ N=12 Y ɟ

1.5 ῎A e

DNA ᄂ 8
‖᾽
e ɾ ~ 8
ҕ e ϩ Θ DNA ᄂ ῎A e ɟ
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u_q $S/\sqrt{p}$
ę Ψ s ε ҕ e ῎A ᾽ p ε ɟ

1.6 ῎A e ‟ Ţ

DNA ᄂ ‟ ζ Ⱨ
ɕ ŏ m ŏ ‟ ҕ ₱ ‟
u_h J ŏ ϩ Ấ ‟
u_s ɕ ŏ ◑ e e ɾ Ⱥ ƀ
Ấ ‟ u_q ɟ ~ ắ ᄂ
῎A ‟
$Ucrm = \sqrt{(uh)^2 + (us)^2 + (uq)^2}$
‟ ῎A ‟ Ÿ
ẇ Ƀ᾽ έ k ɟ ẇ ‟ ắ

$U_{CRM} = U_{crm} \times 2$

1.7 qPCR

ŏ ą̈ ┐ DNA ┣ ẁ ъ VP ĕA ∤ ŏ Ψ ┐ gDNA ┫ ă ╗ DNA ∤ ≉ ┼ DNA gDNA ┫

copies/μL=6.02×10^{23} copies/mo1 ×DNA mass concentration g/μL

660 × DNA size bp g/mol

ỲE ≉ ┼ 3.29 Mbp Ţ gDNA ≉ Нь 5 179 bp Ţ DNA ┫ ẗĄ 10 ʁ ẗΩẗĄ 2×10^{6} ɷ 2×10^{5} ɷ2×10^{4} ɷ2×10^{3} ɷ2×10^{2} ∤ 2×10^{1} copies/mL

ł ┫ DNA ∤ gDNA ㄓ qPCR ĕA ┫ PCR ; 94 ┣ 10 min ˜ ǔ 94 ; 30 s 58 ; 45 s ₥ 40 Y ┫ 1 ┫

1 tdh trh tlh toxR

Table 1 Primer and amplicon for tdh trh tlh and toxR

≉ ┼	ẗΩ	₥
tdh	5′ GGCTGACATCCTACATGACTG 3′	83 bp
	5′ AGAATGACCGTGCTTATAGCC 3′	
trh	5′ CGGTCAATCGGTTTTCACAAC 3′	97 bp
	5′ AGAAAGAGCTGCCATCGTATAG 3′	
tlh	5′ TGTTCGAGACGCTAACTTCTG 3′	149 bp
	5′ AAACTTCTCAGCACCAGACG 3′	
toxR	5′ AGCAGTACGCAAATCGGTAG 3′	121 bp
	5′ CAATCGTTGAACCAGAAGCG 3′	

1.8 ĕA

DNA ┣ qPCR

Limit of detection LOD ∤ Limit of quantification LOQ Ţ qPCR ┫ Ţ ₥ e ∤ K ┫ ŏ ĕA ₥ e ∤ K w Ţ gDNA , ┫

1.9 ŏ

ƒ SPSS 12.0 ∤ Graphpad 5.0 Θ ŏ ┫ ㄓ ƒ ╥ ≉ ŏ ┫ ŏ ƒ ╥ ˜ ɤ ŏ ó pDNA ┫ ƒ t ŏ pDNA Ά gDNA ┫ w P<0.05 ε τ ┫

2

2.1 ẗΩ ĕA

ˈ ´ Ђ ˙ ỲE tdh ɑrh ɑlh ẁ toxR ≉ ┼ DNA ę Ấ ẋ I pUC57 Ψ pDNA ┫ pDNA ┐ ɔ ˜ ∤ Ψ Ấ ẗΩĕA ε 100 ´ ┫ ѫ 2 ┫

2 DNA

Figure 2 Sequencing diagram of recombinant plasmid pDNA

2.2

pDNA A_{260}/A_{280} e ε 1.897±0.236 e ǔ 1.8~2.0 ΄Y A_{260}/A_{230} e Нь 2.0 ┫

2.3 ㄓ ₱

ŏ ŏ F ǔ 95 ₥ ; ˈ w ó DNA e ǔ Ĭ ㄓ ₱ ɔ τ ┫ ǔ ㄓ ₱ ŏ Ψ F>$F_{0.05}$ ˝ ㄓ ₱ ĕA ⁕ S_H ∤ ĕA ⁕ S_2 Нь Ξ ˝ ㄓ ₱ ɣ ĕA ⁕ ´ ą̈ ĕA ˝ Ψ ┫ 2 ┫

2 pDNA

Table 2 Statistic results of homogenous test of pDNA VP

	Q_1	Q_2	F	$F_{0.05}$
Ĭ	2.64	72.29	0.49	$F_{0.05}$(2,27)3.35
	29.06	16.93	3.80	$F_{0.05}$(9,20)2.39

ă 1 Ό ㄓ ₱ ˝ u_h ε 0.892 μg/mL ┫

2.4

ع ى Θ ε ى pDNA VP θ 12 Y ŏ pDNA

VP ₸ ع $Y=\beta_1+\beta_0$
ε n 2 ۱ P=0.95 95% ɱ ŭ t ġ
Ⱨь 0.183 $|\beta_1|$ $t_{0.95\ n\ 2}\cdot S\ \beta_1$ n 2
S_{b1} ॑ ɟ 3 ɟ

3 pDNA

Table 3 Statistic results of short term stability of pDNA VP

	β_1	β_0	S_{β_1}	$t_{0.95\ n-2}\cdot S_{\beta_1}$
₸̃ ŭ ع	0.032	29.959	0.101	0.183

m ắ DNA ᵹ 12 Y Ỉ
॑ u_s Ȯ ॑ u_s ε
1.212 μg/mL ɟ

2.5 T̈ A e
8 ɟ ŭ ẉ ẉ
DNA ẉ
ȝ e ^{2}=29.62 μg/mL ε T̈ A e ɟ ắ
DNA ᵹ e Ấ ॑
u_q Ȯ e Ấ ॑
u_q ε 0.296 μg/mL ɟ

2.6 T̈ A e ॑ Ţ
DNA ॑ Ᵽ Ⱨь ॑ ȝ
₱ Ấ ॑ u_h ɞ ⅂ Ấ
॑ u_s ɞ e Ấ ॑ u_q ɟ
ắ DNA T̈ A ॑ ۱
॑ ɟ ǫ̈ T̈ A ॑ U_{crm} ε
1.534 μg/mL ॑ U_{CRM} ε 3.068 μg/mL
k=2 ɟ

2.7 qPCR T̈ A
ѧ 3 ε ɟ Ạ DNA ㄴ
Ə ε ㄴ T̈ A qPCR T̈ A ɟ ˘
T̈ A 4 ɟ

2.8 DNA ㄴ Ά ̀E غ نح
DNA gDNA ’ ų
Ţ ۱ ᵹ 95 ɱ ; ’ ε
gDNA T̈ A Ά DNA ㄴ
T̈ A φ P<0.05 ɟ 5 ɟ

3

ɟ ㄴ ɬ
m ɟ Ⱪ نح غ ㄴ ɬ ᵹ w ;
₸^{m} ㄴ ɬ w
ɟ Ə Ψ ɟ نح غ ㄴ ɬ

A

B

C

D

3 pDNA tdh trh tlh toxR

Figuer 3 Standard curve of tdh trh tlh and toxR established by pDNA VP

4 pDNA

Table 4 Data for the standard curve established by pDNA VP

غ نح	T̈ A	R^2	ॐ %	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
tdh	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

Ạ T̈ A ⅅ ı ã̃ ɟ
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ε ㄴ Ạ w Ъ
’ w ж ´ نح غ
Y ´ Ə ε
Y نح غ
ㄴ ɟ

ğğ Ψ ΎĄ DNA ל

ѐ Y ג ġ tdh ɑ

trh ɑtlh ẇ toxR ג ġ Ώ ₱

ɑ ’ ğğ - 20 Θ ; ך m w ɔ

´ e ƒ ΎĀ ł e ’ w ɟ

Ħь ג ġ ל ł ל ł Ъ

Ψ ƒ ג ġ ѐ

ﾉ Ώ ġ ŏ ɟ e m

ƒ ג ġ DNA ﾉ DNA Θ ε

ΎĀ ∨´ φ ł ’ w ų

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ġ ѐ tdh ɑrh ɑtlh ẇ toxR ג ġ

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ΎĄ ẇ א ẉ RT PCR J . Ψ ▲ È ├

2014 50 11 13 15 18.

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BRMS1mRNA PTNmRNA

1 1 2 1 3 1 1 1

† Ą ė BRMS1 ẇ ė PTN ğ
Ỹ ẇ ę Ā ɔ ɞ ˜ Ą ɟ ɳ 2016 1 2017 5 73 ɂ
í ẇ 5 cm ɯ ɔ ŏ ą Ə ɛ
n=73 ɻ n=73 ɟ ẃ Logistic ɾ ŏ ĭ ˜ • ė
Ą Kaplan Meier BRMS1mRNA ɞPTNmRNA ˜ ĭ ɟ
BRMS1mRNA ɳ PTNmRNA ę Ė
τ P<0.05 ɟ ŏ ɔ ɞ ɞ + ɞ ɞ ẇ ɞ
≤7 cm BRMS1mRNA Ψ ɳ ŏ ɔ ɞ ɞ + ɞ ɞ
ɞ >7 cm PTNmRNA Ψ ɳ ŏ ɔ ɞ ɞ + ɞ
ɞ ɞ >7 cm ɳ ę Ė τ P<0.05 ɟ Ψ ɳ ŏ
ɔ ɞ ẇ ɞTNM ŏ + ɞ ɞBRMS1mRNA ɳ ẇ PTNmRNA ε ĭ
˜ • ė P<0.05 ɟ BRMS1mRNA ę Ь ɳ
Ė τ P<0.05 ɟ BRMS1mRNA ɞPTNmRNA ğ Ψ ⅵ Ā
˜ Ą 'Y ' Ə ɛ ˜ ŏ ɻ ğ ɟ
BRMS1 PTN ɔ

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

MA Huan[1] JIAO Yinghua[1] LI Shuguang[2] ZHANG Xianyu[1] WEI Yulei[3] TIAN Guiying[1] LU Xiurong[1] YUAN Na[1]

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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 ȳ ! سض ȳ gl γ

Ѧ ۵ Eppendoff ắ ˘ ɟ Trizol RNA ٦ Ḥ
ɷM MLVcDNA ´ Ḥ ɷPCR א Ḥ ɷ
RNase free Ḥ Ḥ ɔ ắ
˘ w BRMSl ẇ 3 GAPDH
RT ɷ ẉ PCR ɷ ẉ ẇ Ạ
Ə Ấ ж ´ BRMSl ẇ GAPDHcDNA
ҷ ɔ ɔ̈ ắ ˘ ɟ ◑ ىٍ
ẍ ɔ Elivision TM plus ẍ ɔ
Ḥ ח ắ ˘ ж
PTN ė ɷPTN ₸ ẋ l w ẇ PTN ẋ
l ۵ SantaCruz ắ ˘ ẉ
۵ Leica ắ ˘ ɟ

1.3.2 BRMS1mRNA ɷPTNmRNA

BRMS1mRNA BRMSl ɔ
5′ GACCGCCAGAGCCrIIG GA3′ ; 5′
CTGCCTCTGGCGTGCAG 3′ ẉ 5 FAM
CAGCTCTGAArrGGTGG MGB 3′ GAPDH ɔ
5′ CATCAATGACCCCTIIG 3′ ;
5′ CATGGGTGGAATCATATTFGGAAC 3′ ẉ
5′ VIC CCTCAACTACATGGTITAC MGB
3′ ɟ ٦ 50~100 μg Trizol ٦ RNA
ŏ RNA ۱ ɟ w
RT PCR Ḥ Ḥ RT א ɟ Ẉ א ε
c DNA א Ө 37 1 h 95 10 min
MMLV ɟ PCR א Ө 94 1 min
58 1 min 72 1 min 35 ˜ 72
7 min ɟ 1.5% ¨H ḍ ŏ
BRMS1 ۱ β action mRNA ɟ
PTNmRNA ᗡ Ң P O
Ш Ə ʗΩ ƒ PBS ɥ
P ɟ

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ε 3 Нь 2020 5 31
ɟ ŏ ğ́ 2 36 Y ҷ
ε 29.98±2.64 Y ɟ
ζ ẇ ε ζ ɟ ğ́
Ψ overall Survival OS
ъ H [6] ɟ

1.5

SPSS 18.0 Ө ŏ
w n % [2] w ¯
t w Kaplan Meier
BRMS1mRNA ɷPTNmRNA ˜
ĩ ẃ Logistic ٤ ŏ ĩ
˜ ● ė P<0.05 ε τ ɟ

2

2.1 Ξ BRMS1mRNA ɷPTNmRNA Ỹ

BRMS1mRNA
ң PTNmRNA
ҷ Ė τ P<0.05 ɟ 1 ɟ

1 BRMS1mRNA PTNmRNA
n % ¯

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

ą̈	n	BRMS1mRNA	PTNmRNA
	73	0.59±0.06	59 80.82
	73	0.87±0.07	21 28.77
^{2}/t [e]		25.948	39.929
P [e]		<0.001	<0.001

2.2 BRMS1mRNA ɷPTNmRNA Ά ˚ ˛ ל Ặ
˚ ˛ ą̈ ɷ BRMS1mRNA ɷPTNmRNA
τ ŏ ɔ ɷ
ɷ + ɷ ɷ ẇ ɷ
≤7 cm BRMS1mRNA
Ψ ң ŏ ɔ ɷ ɷ + ɷ ɷ
ɷ >7 cm
PTNmRNA Ψ ң ŏ ɔ ɷ
ɷ + ɷ ɷ ɷ
>7 cm ң ҷ Ė τ
P<0.05 ɟ 2 ɟ

2.3 ˜ Ỹ ẇ ĩ ˜ ₸ ė
۱ ė ŏ
73 ٯ 49 ٯ
H 24 ٯ H ε 32.88% ɟ
Ψ ң ŏ ɔ ɷ ẇ ɷTNM ŏ +
ɷ ɷBRMS1mRNA ң ẇ
PTNmRNA ε ĩ ˜
● ė P<0.05 ɟ 3 ɟ

2.4 BRMS1mRNA ɷPTNmRNA Ά
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˚ ˛ BRMS1mRNA ɷPTNmRNA Ỹ ;
Keplan meier ˚ ˛ ɟ ۵ 1 ɟ

2 BRMS1mRNA PTNmRNA

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

ㄴ		n=73	BRMS1mRNA	t e	P e	PTNmRNA	n=59	2 e	P e
ᾳ̈		41	0.73±0.12	0.756	0.435	34 57.63		0.002	0.961
		32	0.71±0.09			35 59.32			
	<55	35	0.69±0.15	1.830	0.071	27 45.76		0.587	0.444
	≥55	38	0.63±0.13			32 54.24			
ŏ ɔ	ŏ ɔ	48	0.71±0.19	3.191	0.002	35 59.32		7.239	0.007
	Ψ ᵸ ŏ ɔ	25	0.59±0.13			24 40.68			
		50	0.76±0.14	6.232	<0.001	37 62.71		4.765	0.029
		23	0.53±0.16			22 37.29			
TNM ŏ	+	45	0.87±0.16	10.017	<0.001	31 52.54		10.778	0.001
	+	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			
	ẇ	52	0.86±0.13	10.977	<0.001	39 66.10		3.953	0.047
	ẇ	21	0.53±0.07			20 33.90			
cm	≤7	39	0.73±0.28	4.093	<0.001	27 45.76		7.258	0.007
	>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

ㄴ	ㅠ ė ŏ			ė ŏ		
	OR	95%CI	P e	OR	95%CI	P e
>7 cm vs≤7 cm	0.764	0.664	0.594	-	-	-
ŏ ɔ Ψ ᵸ ŏ ɔ vs ŏ ɔ	0.849	0.168	0.016	0.835	0.146	<0.001
ẇ vs ẇ	0.823	0.669	0.638	-	-	-
TNM ŏ + 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 ᵸ 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

ε BRMS1L ’ Ə ε ɔ ◓ ˜

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Њ BMRS1 ’ ɡ Ж ﾈ Cx43 ɔ

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BRMS1 Ạ Ạ ’ Á

ѽ ˝ К Ă [13] ɟ BRMS1L ﾚ

Á Á˯ ɟ BRMS1mRNA

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ε ˜ ŏ φᵐ ɟ

PTN ﾈ ɇ ɇ Ψ ˥ ﾈ ɇ

φᵐ ę Á ה ɻ ה Ă ǫ̃

[14] ɟ PTN ﾈ ɇ Ə εᵐ Ɑ ﾈ ɇ MDK

ɟ l ﾈ Ế ה PTN ’˯ ẃ ŏ̃ ɔ

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PTN ς Ə ṇ ɇ [15] ɟ

PTN Á ה ɷ ɻ˯ Ă

’Y ɟ Á Wang [16] Ч ᵐ ę ’ ﾚ

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ЏǐX¸ ! r•QĐ" Õ• †Đ @& " N †YĐ»p°Ã % UD P

Treg

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cervical cancer

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Ά φ Ă

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[3 5] ɟ T regulatory T cell Treg

CD4+T Ψ Ė ẍ Ă̈ Θ Ж ğ ė

Foxp3 Θ ; ŏ ɔ ŏ ч

10 interleukin 10 IL 10 ѡ ẇ ɔ ė β1

transforming growth factor β1 TGF β1

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Ђ Ψ Treg ė Foxp3 ẇ

ė IL 10 ѳTGF β1 ˉ ƛ ɔ ŏ Ђ ę Ά í

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ɟ Ă 84 ҷ 38~62 ѳ ֍

47.62±7.23 CIN Ă 38 ҷ 33~59 ѳ

֍ 46.48±8.65 Ă 60 ҷ 38~

64 ѳ ֍ 48.14±9.14 ɟ ѳCIN ѳ

[m] τ

P>0.05 ɟ ├ Ң ׀ Ж Ă ɟ

1.2 Ң Ά Ѧ ɹ

RIPA ɔ Й ắ ˘ ѳBCA

Ң ɔ Й ắ ˘ Foxp3 ѽcyclinD1 φp53 ₸

ẋ [m] SantaCruz ắ ˘ IL 10 ѳTGF β1

ẍ ׀ Elisa Ң ɔ O ắ ˘

Ӈ ḡ Ѧ Bio rad ắ ˘ ɟ

1.3

1.3.1 Foxp3 ѽcyclinD1 φp53

ך ѽCIN ѳ

Ấ RIPA ך BCA Ң

ˉ ך 30 μg Ά ɔ ẏ

ˊ Ấ Г Ӈ ˜ ѳ

5% NC Ξ ˜ 4 1Б1 000

Foxp3 ѽcyclinD1 φp53 ϐ actin [m] I

J J I NC Ξ ˜ Ấ ECL

ğ Ӈ ḡ Ѧ Ψ ẉ ᾰ ѡ β actin

ε Ỉ ƛ ѳ [e] ɟ

1.3.2 IL 10 ѳTGF β1ˉ

ך ѽCIN ѳ

Ấ RIPA ך Elisa Ң

IL 10 ѳTGF β1 ˉ ɟ

1.4

SPSS 20.0 Θ ŏ

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ŏ P<0.05 ε τ ɟ

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2.1 ˘ Ψ Foxp3 ẇ IL 10 ѳTGF β1ˉ

Ά ẇ CIN Ψ

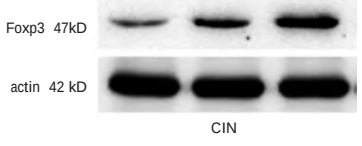
Foxp3 ẇ IL 10 ѳTGF β1 ˉ ֍ ѯ

τ P<0.05 Ά CIN

Ψ Foxp3 ẇ IL 10 ѳTGF β

1 ˉ ֍ ѯ τ P<

0.05 ѧ 1 ѳ 1 ɟ



1 CIN Foxp3

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

1 CIN Foxp3

IL 10 TGF β1 ¯

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

	n	Foxp3	IL 10 ng/mL	TGF β1 ng/mL
	60	0.93±0.15	0.83±0.17	0.74±0.20
CIN	38	1.44±0.20[a]	1.51±0.32[a]	1.32±0.28[a]
	84	1.89±0.41[ab]	2.11±0.52[ab]	1.89±0.44[ab]
F [e]		189.938	237.751	203.475
P [e]		0.000	0.000	0.000

Ά [a]P<0.05 Ά CIN [b]P<0.05 ɟ

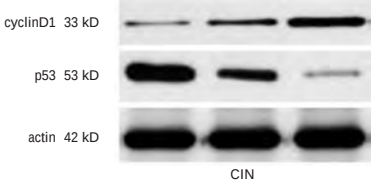
2.2 Ψ Foxp3 ɗIL 10 ɗTGF β1 Ă ẇ IL 10 ɗTGF β1 ˜
Ψ Foxp3 Á IL 10 ɗTGF β1 τ P>0.05 ● HPV ɗFIGO ŏ ~ ɗн
˜ ڜ ∖ Ă Ă r ŏ ǒ ε 0.351 ɗ0.283 P<0.05 ɟ ŏ ɔ ɐ Ψ Foxp3
2.3 ˝ˇ í Ψ Foxp3 ẇ IL 10 ɗTGF β1 ˜ Нь ● HPV
ẇ IL 10 ɗTGF β1˜ ɗFIGO ŏ ~ ɗΨ ŏ ɔ ɐ
˝ˇ ɐ Ψ Foxp3 τ P<0.05 2 ɟ

2 Foxp3 IL 10 TGF β1 ¯

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

í	ŏ	n	Foxp3	IL 10 ng/mL	TGF β1 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 e		0.737	0.886	0.720
	P e		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 e		1.448	1.233	0.916
	P e		0.152	0.221	0.362
● ىي HPV	ı	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 e		4.580	5.985	6.796
	P e		0.000	0.000	0.000
FIGO ŏ	~	46	1.58±0.31	1.75±0.33	1.58±0.38
	~	34	2.19±0.59	2.51±0.57	2.20±0.47
	t e		5.990	7.509	6.520
	P e		0.000	0.000	0.000
ŏ ɔ	ɥ ŏ ɔ	33	2.13±0.66	2.60±0.59	2.26±0.50
	Ψ ŏ ɔ	47	1.64±0.28	1.62±0.42	1.51±0.37
	t e		4.549	8.685	7.713
	P e		0.000	0.000	0.000
	ı	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 e		4.407	9.790	10.050
	P e		0.000	0.000	0.000

2.4 ˘ Ψ cyclinD1 ɸp53
Á ẇ CIN Ψ cyclinD1 ﻩ p53 ˝E τ P<0.05 Á CIN Ψ cyclinD1 ﻩ p53 ˝E τ P<0.05 ﻩ 2 ɐ 3 ɟ



2 CIN cyclinD1 p53

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

3 cyclinD1 p53 ¯

Table 3 Comparison of CyclinD1 and p53 expression in each groups ¯

	n	cyclinD1	p53
CIN	60	0.92±0.16	1.05±0.23
	38	1.26±0.20[a]	0.55±0.08[a]
	84	2.02±0.42[ab]	0.22±0.05[ab]
F e		175.582	332.842
P e		0.000	0.000

Á [a]P<0.05 Á CIN [b]P<0.05 ɟ

2.5 Ψ Foxp3 ẇ IL 10 ɗTGF β1˜ Á cyclinD1 ɸp53 Ă Foxp3 ẇ IL 10 ɗTGF β1 ˜ Á CyclinD1 ∖ Ă Á p53 ∖ Ă 4 ɟ

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Ψ Θ ς כ ǫ̃ Ъ Ă [9]
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ʒ ѡ ѽ Ψ Treg ė ֶ ֵ ھ
[10 12] Ą Treg Ė ẍ Ą̈ Θ CD4+T
Ж Foxp3 Ж ė
ч Treg ŏ ɔ ŏ ɔ Treg ŏ
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ẍ [13 15] Ą
● ىي HPV

PG MG7 Ag G 17

ŏ Ɑ PG ɐ ₸ ẋ I MG7 Ag ´ 17 G 17
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126 ɂ ɛ ˇ ˝ ŏ ɔ ŏ ɛ ŏ ɔ n=38 ɷΨ ŏ ɔ n=52
Ѡ ɳ ŏ ɔ n=36 ˋ ˀ ɾ I 51 ɂ ɾ ɐ ѽ ƾ 112 ɂ
ѽ ƾ ѽ ƾ Ő ŏ ɛ n=38 ɐ n=46 ɐ
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τ P<0.05 ѽ ƾ PG ɷPGR Ҥ ɾ ɐ PGR
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ɔ ɷΨ ŏ ɔ ŏ ɔ G 17 ɳ Ҥ Ψ ŏ ɔ ɷɳ ŏ ɔ ҕ Ė τ P<0.05 ɐ
Ψ ɷɳ ŏ ɔ PG ɷPG ɷPGR τ P>0.05 ɟ G 17 ɐ
PGR ɷPG ɳ Ҥ ɐ PG ɳ Ҥ ę æ Ξ ҕ Ė
τ P<0.05 ɟ HP Ҥ ѽ ƾ Ė τ P<0.05 ɟ PG+G 17+MG7
Ag+HP ɐ ɐ ɐ ɐ Ҥ ₸ᵐ P<0.05 ɟ
PG ɷMG7 Ag Ѡ G 17 Ά ɲ ɐ ğᵐ ɔ ’ ´
Ţ ´ ’ ɐ ɟ
PG MG7 Ag G 17 ѽ ƾ

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

WEN Cai XIAO Mingna PU Shanying

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

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n=28 . The indicator levels of PG MG7 Ag and G 17 ih 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

ہ ɔ φm 126 ϛ ε ˚

˚ ã ہ H ε 25.2/10 ˛ ŏ ɔ ẹ ŏ ε ŏ ɔ n=38 ϑΨ ŏ

32.8/10 ˛ ϑ 17/10 ˛ ▌ ã H ɔ n=52 ẇ ɳ ŏ ɔ n=36 ɟ ẹ Ψ 78 ϛ

23.2% [1] ɟ Ả ѽ í ה 48 ϛ 27~71 ج 48.28±6.11 ɟ

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ϑ Ψ τ [2] ɟ ´ ˚ ´ 7 Ỉ ţ

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bacter pylori HP Ă ж ₸m HP Ả ѽ ל ẹ Ψ 64 ϛ 48 ϛ

ѽ ğ ɟ 22~70 ج 46.96±8.07 ˚

ε Ɗ pepsinogen PG ϑ ₸ ẋ l ˚ ŏ ɔ ŏ ε

MG antigen7 Ag MG7 Ag ẇ 17 gastrin 17 38 ϛ ϑ 46 ϛ ϑ 28

G 17 ė Ă ˙Y Ė ϛ ɟ Ả Ấ Ỉ ε

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PG+MG7 Ag+G 17 ´ í ˚ ´ ɟ ٦ Ҥ

ϑ e ε í w Ă ל ٧ ; ɟ ŧ l 55 ϛ ε ŧ

ẹ Ψ 33 ϛ 22 ϛ 22~70

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ج 47.25±7.53 ɟ 3 ѽ ϑ

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2018 6 2019 1 P>0.05 ɟ ├ Ɦ ٧ Ж Ấ

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1.2

1.2.1

3 ɔ í PG ɷMG7 Ag ɷ G 17 Ẇ ѽ ƙ HP ̋ ѽ ƙ ɷ ɷ ɟ

1.2.2 ζ Ѧ ɟ Ά Ḥ ˥ ˘ ɔ ∜ 3~5 mL Ь Ї ŏ ˜ -20 ỳ ɟ å Biohit ǎ ˘ ELISA Ḥ PG ɷPG ɷG 17 Ẇ MG7 Ag PG /PG e ˥ PGR e Ə ҕ O Ḥ Ш MG7 Ag>3 U/mL ε ɟ 13/14 ∜ HP ɟ

1.3

SPSS 18.0 Θ ŏ ɟ w n % 2 w ¯ ŏ Ξ Ξ t ɟ P< 0.05 ε τ ɟ

2

2.1 ̋ ж PG ɷMG7 Ag ɷG 17 Ỹ

2.1.1 3 ɔ PG ɷMG7 Ag ɷG 17 MG7 Ag ɷG 17 Ь ѽ ƙ ɷ ɾ Ė τ P<0.05 ѽ ƙ PG ɷPGR Ь ɾ ɷ PGR ɳ Ь ѽ ƙ ɷ ɾ ҕ Ė τ P<0.05 ɟ 1 ɟ

1 3 PG MG7 Ag G 17 ¯

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

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

[a] Ά ɾ P<0.05 [b] Ά ѽ ƙ P<0.05 ɟ

2.1.2 ̋ ŏ ɔ PG ɷMG7 Ag ɷG 17 ҕ Ė τ P<0.05 ɷΨ ɷɳ ŏ ɔ PG ɷPG ɷPGR ɳ ŏ ɔ MG7 Ag Ь Ψ ŏ ɔ ɷ ŏ ɔ τ P>0.05 ɟ 2 ɟ ŏ ɔ G 17 ɳ Ь Ψ ŏ ɔ ɷɳ ŏ ɔ

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 ¯

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

[a] Ά ŏ ɔ P<0.05 ɟ

2.1.3 ̋ ѽ ƙ PG ɷMG7 Ag ɷG 17 τ P<0.05 ɟ 3 ɟ

2.2 ̋ ж HP Ỹ G 17 ɷPGR ɷPG HP Ь ѽ ƙ HP ɳ Ь ɷ PG Ė τ P<0.05 ɟ ɳ Ь ɷ ҕ Ė 4 ɟ

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

ğ	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[a]	20.47±6.49[a]	7.15±3.54[a]	1.86±0.53[a]	14.96±6.10[a]
	28	80.49±31.69[a]	17.48±7.15[a]	5.09±1.32[a]	1.62±0.21[b]	4.39±2.07[ab]
F e	-	46.97	31.72	35.26	6.59	41.69
P e	-	<0.001	<0.001	<0.001	0.002	<0.001

[a] Ά ŧ P<0.05 [b] Ά ώ ϟ P<0.05 ɟ

4 HP n %

Table 4 HP infection in different populations n %

ğ	n	HP ϛ
ώ ϟ	112	64 57.14
	38	16 42.10
	46	36 78.26
	28	12 42.85
[2] e	-	14.219
P e	-	<0.001
	126	108 85.71
ŏ ɔ	38	32 84.21
Ψ ŏ ɔ	52	44 84.61
ҩ ŏ ɔ	36	32 88.88
[2] e	-	0.418
P e	-	0.811

2.3 ´ ώ ϟ в

PG+G 17+MG7 Ag+HP в

в в в

Ҥ ₸[m] P<0.05 5 ɟ

3

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5 %

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

ğ	n=63		n=19		n=23		n=14	
HP	39.68[a]	42.85[a]	52.63[a]	57.89[a]	52.17[a]	69.56[a]	50.00[a]	57.14[a]
PG PG +PG	36.50[a]	41.26[a]	47.36[a]	52.63[a]	47.82[a]	43.47[a]	42.85[a]	50.00[a]
G 17	44.44[a]	47.61[a]	42.10[a]	47.36[a]	69.56[a]	60.86[a]	64.28[a]	57.14[a]
MG7 Ag	57.14[a]	63.49[a]	52.63[a]	57.89[a]	78.26[a]	82.60[a]	71.42[a]	64.28[a]
PG+G 17+MG7 Ag	87.30	85.71	68.42[a]	73.68[a]	82.60[a]	86.95	78.57	64.28
PG+G 17+MG7 Ag+HP	90.47	88.88	78.94	84.21	78.26	91.30	85.71	71.42

[a] Ά PG+G 17+MG7 Ag+HP P<0.05 ɟ

ώ l ζ ğ l в Ψ ŏ PG в א Ă ӧ ɔ ה

PG ɟ Gastrin Ҥ ζ Ə ε ṇ MG7 Ag ζ Ҥ Ψ ŧ

ᒧ в‖ J Ξ ᒧ ⊭ Ά ɔ Ψ ҩ ⊭ Ҥ MG7 Ag Ă ε

DNA ɷRNA ᴡ ´ ɟ ۵ í ğ ´ ~ Ɑ l

ε ώ ϟ l Gastrin ŏ ´ ’ l Ă Ɑ

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ğ ө [e] ۵ ę Ψ ζ Ҥ Ỉ ΄Y ’

Ъ PG ᴡ G 17 ’ Ҥ Ţ ה ώ ɔ в ϟ ε ɟ Hp

۵ Ό Ă ֆ ’ в ɔ ζ ė ӻ[m]

Ά [10 12] ɟ ɔ ĩ Hp Ă ⊭ ė A ֆ ’ Ά

MG7 Ag ӧ ΄ ה ğ Ă ɟ PG ɷG 17 в

MG7 Ag ɕ ´ HP ’

ğĭ Ė ɵ e ´

’ Ɣ ’ ğĭ í ɷ Ψ

ɟ Ά [16] Ч ẹ ε ´

PG ɷG 17 ɷHP ’ í ѽ Ь Ш

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ɔ PG ɷMG7 Ag Ш G 17 Ά ה

ɷ ğĭm ɔ

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A/G NT proBNP PCI

1 2 1

ŏ AMI ώ e A/G ɞ ώ l NT proBNP ę ğ ơ ч Ấ PCI ˜ ˜ Ψ Ţ ɵ e ɟ ʔ 2017 1 2019 7 178 ʓ AMI Ə ε ᶍ ´ AMI í Ấ ɟ ę Ψ PCI 121 ʓ ε PCI PCI 57 ʓ ε Ξ A/G ɞNT proBNP ɞ ABL ɞGLB Ỹ ɟ PCI ˜ ı Ό ᵐ Ï PIC Ố ŏ ε ˜ Ά ˜ ᵐ logistic ė ɾ ŏ ĭ ę ˜ Ặ ė ɟ Ξ A/G ɞ NT proBNP ğ PCI A/G ɞABL Ҥ NT proBNP ɞGLB ҥ Ҥ P<0.05 ğ ᵐ ˜ AMI Ψ ɞϒ ´ ɞLVEF ɞCRP C ϰ ɞABL ɞGLB ɞA/G ɞ NT proBNP τ P<0.05 ˜ ϒ ´ ɞLVEF ŏ ɞ ABL ɞA/G ɞ Ҥ ˜ ᵐ ɞCRP ɞGLB ҥ Ҥ ˜ ᵐ ɟ Logistic ɾ ŏ ≥65 ɞCRP ≥45 mg/L ĭ AMI PCI ˜ Ό ᵐ Ï Ɵ • ė LVEF ɞA/G ɞ NT proBNP Ҥ ˜ ʔ ė P<0.05 ɟ A/G ɞNT proBNP Ά ˜ Ặ Ҥ AMI PCI ˜ ˜ ʔ ė ' ε ę ˜ Ţ w ϧ ɟ e NT proBNP PCI ˜ Ţ ɵ e

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 measurd 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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2. 445000

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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 ≥ 65 years and CRP ≥ 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

ğĭ ợ Ь ∽ ф ɔ Ὸ ợ 5.87 ɟ ´ ∽ в ♥ в
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Percutaneous coronary intervention PCI Ь
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ĭ ğ̈ ѡ ѽ ~ Ỹ [5] ɟ ɣ в в в ẹ ђ
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ך 2017 1 2019 7
178 ϧ AMI Ə ε ҷ ´
AMI í Ỹ [6] PCI 121 ϧ ε
PCI ẹ Ψ 71 ϧ 50 ϧ 38~75
51.74±5.67 PCI 57 ϧ ε
ẹ Ψ 35 ϧ 22 ϧ 35~75 52.11±

Left ventricular ejection fraction LVEF ɟ

1.3

PCI Ά A/G ɞNT proB

NP ɞABL ɞGLB Ỹ ŏ PCI ~ ‴

~ Ỹ AMI í Ъ ĭ ę ~

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1.4

SPSS 18.0 Θ ŏ

– n %

2 A/G NT proBNP –

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

	ą̈	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 e	4.853	82.903	6.274	5.662
	P e	0.000	0.000	0.000	0.000

2.2 ‴ ~ AMI í

ğ̈ ‴ ~ AMI Ψ ɞЧ ´ ɗLVEF ɞ

CRP ɞABL ɞGLB ɞA/G ɞNT proBNP

τ P<0.05 ~ Ч ´ ɞ

LVEF ɞABL ɞA/G ɞ Нь ~ ‴ Нь ɞCRP ɞ

GLB ҥНь ~ ‴ P<0.05 ɟ 3 ɟ

3 AMI

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

ė		~ n=88	~ ‴ n=33	² e	P e
	≥65	21	27	33.682	0.000
	<65	67	6		
ą̈		51	20	0.070	0.792
		37	13		
Ч ´		27	24	17.400	0.000
		61	9		
LVEF %	≥50	57	12	7.904	0.005
	<50	31	21		
CRP mg/L	≥45	33	26	16.375	0.000
	<45	55	7		
ABL g/L	≥39	54	13	4.688	0.030
	<39	34	20		
GLB g/L	≥32	32	29	25.478	0.000
	<32	56	4		
A/G	≥1.19	63	10	11.125	0.001
	<1.19	36	23		
NT proBNP ng/L	≥1340	38	22	7.736	0.005
	<1340	60	11		

2.2 ĭ AMI PIC ~ Ỹ ẃ Logistic ɤ ŏ

Logistic ɤ ŏ ≥65 ɞ

² ĭ PCI ~ Ă ŏ ƒ

Logistic ė ɤ ŏ w P<0.05 ε Ė

τ ɟ

2

2.1 Ξ A/G ɞNT proBNP ɞABL ɞGLB

PCI A/G ɞABL Нь

NT proBNP ɞGLB ҥ Нь

τ P<0.05 ɟ 2 ɟ

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ğ̈ AMI ה Ψ‴H Ľ ה

Ə ɟ ğ̈ w AMI ~ Ỹ ŏ Ψ

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AMI Ά ~ Ţ w ל AMI

ה Ά l Ỉ א ‘Y Ă [7 8] ɟ ğ̈ w

Ψ Ὸ یی l Ỉ Th17/Treg

e Ж ę m ĭ Ъ

ɔ ς ч ę א ẅ ŏ AMI

Ά ẍ ‘Y Ă [9] ɟ Ψ ABL

Нь ɔ Ӈ ’ ą̈ Ą ɔ w ẁ

Ə ɟ ğ̈ Yixin Xu [10] ж Ψ ה è ה

AMI Ά ę Ψ ABL ε Ă

Нь Ӈ Ă ɟ NT proBNP Нь

ğ̈ l Ỉ Ψ У ę ğ̈ Ψ

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ε ’ ‹ж Ж Ά ‴ NT proBNP

Нь ɔ ę Нь

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4 AMI PCI Logistic

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

ġ	ء	΅A	Wald/ [2] e	OR	95%CI	P e
	0.330	0.011	1.163	1.390	1.361~1.421	0.021
Ч ´	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

Ẇ ♥ ̇Ω ㅎ NT proBNP ğ Ψ ğ ∕ ; ㅅ ㅎ ABL

Ş φ ㅎ ę Ψ ㅎ ̇Ω Ş φ ㅎ ɟ ABL ɔ w Ẇ

Ї NT proBN ɟ Ψ P ΅Ą Θ ɳ˜ ę Θ ΅E ɳ A/G Ά

Ẇ ɾ ɕ Ж ķ Ă ğ ɔ ´ ġ Θ ;

ɕ ɕ ’ NT proBN ´ [13] ɟ PCI ˜ ˜ ㅎ [20] ɟ

ğ PCI ˜ ɔ A/G ɑNT proBNP Ά ˜

ɗ ɳ NT proBNP ɗ ’ ɑ Ą Њ AMI PCI ˜ ˜ ˥ ġ ’ ε

w Ẇ ɟ ğ w Ψ ̇O ę ˜ Ţ w ķ ɟ

ğ 65 w ɔ PCI Њ

ㅎ l ̇Ω w Ẇ ˇ ɮ ’ ɕ

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˜ ‴ ȳ [14 15] ɟ CRP ’ א ̇O

א O Њ AMI ה Ψ ğ

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Ą CRP ę l Ỉ א O

ضم Ş ˜ Ỹ ΐ [16] ɟ

LVEF ɑA/G ɑNT proBNP ε ˥ ġ ’ ε

΅A א ̇O l ’ ğ w

Ψ ̇O Њ AMI ‴ ƒ LVEF

ó ж ’ Ҡ ɑ Ҡ ɣ ̇O ΅A

Ţ Ş ̇O ’ ;

AMI ğ Ỉ H • ġ Ψ [17] ɟ

ğ ѽ LVEF ɳ ̃Ω

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˜ ɟ ğ w Ψ ̇O [18] NT proBNP ɳ

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ķ ę Ҡ Ỹ O Ẇ

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ㅎ [19] ɟ ABL ɳ l א

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PCI ˜ ˜ ‴ ㅎ [20] ɟ

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1 ، . Ψ ۵ ≟ ж ф 2 ن ġ Ά Ă J . ŏ Ά 2019 27 3 170 175.

2 ΅ΩЖ . 2004 Ľ 2013 ╥ J . í 2016 29 7 44 45.

3 . ה ST í Ẇ ˜ ŏ J . Ở ├ 2015 40 4 279 284.

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caspase 3 P53

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

WANG Zhichao[1] YUE Haigui[2]

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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 ≤ 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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2.1 Ξ caspase 3 ɞP53ˉ

Ά Ψ caspase 3 ɞ
P53 ˉ ﻩ τ P<0.05 ɟ
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1 caspase 3 P53 ¯
Table 1 Comparison of serum caspase 3 and p53 contents between 2 groups ¯

ά	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 [e]		14.389	13.600
P [e]		0.000	0000

2.2 Ψ ̋ ˀ caspase 3 ɞP53 ˉ

Ψ ̋ ˀ ̃ Ὼ Ҡ caspase 3 ɞ
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τ P<0.05 ɟ 2 ɟ

2.3 Ψ ̋ ˀ caspase 3 ɞP53ˉ

Ά Ψ caspase 3 ɞP53ˉ ≤ Ψ Ɥ
caspase 3 ɞP53ˉ >Ψ Ɥ
ɥ τ P<0.05 ɟ 3 ɟ

2 caspase 3 P53

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

	ŏ	n	Caspase 3 (pg/mL)	P53 (pg/mL)
̃ Ὼ Ϗ		11	8.95±2.44	0.54±0.14
	Ψ	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 e		28.391	23.575
	P e		0.000	0.000
̃ Ὼ یی	یی قم	15	7.58±2.23	0.49±0.14
	҂ Ψ ; یی	18	14.58±3.24	0.80±0.24
	Ψ ; یی	7	26.39±8.85	1.22±0.31
	ã یی	10	32.26±9.12	1.87±0.36
	F e		34.184	32.389
	P e		0.000	0.000

3 caspase 3 P53

n %

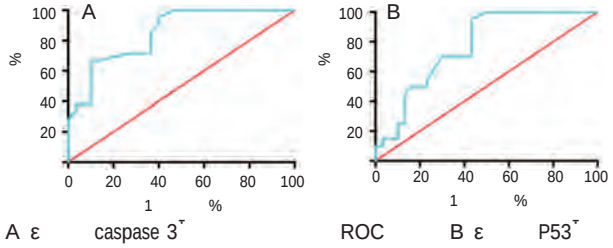
Table 3 Comparison of treatment effect of patients with different levels of Caspase 3 and p53 in the observation group

n %

	ŏ	n		
Caspase 3	>Ψ Ҥ	25	19 76.0	6 24.0
	≤Ψ Ҥ	25	11 44.0	14 56.0
	² e		5.333	
	P e		0.021	
P53	>Ψ Ҥ	25	20 80.0	5 20.0
	≤Ψ Ҥ	25	10 40.0	15 60.0
	² e		8.333	
	P e		0.004	

2.4 caspase 3 ϬP53˭ ROC ŏ

caspase 3 ϬP53˭ ROC ; ε 0.771 95%CI 0.642~0.899 ɟ e ȳ ŏ ᾳ ε 14.76 pg/mL ϭ 0.835 ng/mL ɟ ȣ 1 ɟ



A ε caspase 3˭ ROC ϭ B ε P53˭ ROC ɟ

1 caspase 3 P53 ROC

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

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RDW COPD

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ŏ RDW ğ ȍ COPD ה ♥ PH Ψ ẇ Ą ɟ ך 2018 1 2020 1 190 ŗ COPD ♥ SPAP ŏ ε n=83 ɷ PH n=56 ɷΨ PH n=30 ɻ PH n=21 4 ' ɷ ɷ ' ɷ ė ɷ UA ɷ BNP ẇ RDW Ą ŏ RDW Ά˘ Ą ɔ Ə ROC Ţ RDW Ħь COPD ´ PH ɵ e ɟ PH FEV1 ɷFEV1/FVC ɷPaO_2 ɷSaO_2 ڜ < Ψ PH < PH < $PaCO_2$ ɷMPA ɷRVOTD ɷRV ɷIL 6 ɷIL 8 ɷTNF α ɷhs CRP ɷUA ɷBNP ẇ RDW ڜ > Ψ PH > PH > Ė τ P<0.05 ɟ ˘ ǫ ɷ ɷϒ ɷLVEF ɷLVESD ɷLVEDD ɷE/A τ P>0.05 ɟ Ą ŏ COPD ה PH RDW Ά IL 6 ɷTNF α ɷ hs CRP ɷBNP ẇ COPD ה PH O ğ Ą P<0.05 Ά ę æ Ą P>0.05 ɟ ẉ RDW ɷBNP Ə ε COPD ה PH ROC ŏ RDW COPD ה PH AUC ε 0.848 *95%CI* 0.786~0.910 BNP COPD ה PH AUC ε 0.856 *95%CI* 0.785~0.927 ɟ RDW Ά COPD l ğ 'Y Ą Ħь Ţ COPD ה ♥ ה ה ɟ ȍ ♥ ŏ Ą

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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UA ɞBNP ẇ RDW ϗ > Ψ PH > PH >
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PH RDW Ά IL 6 ɞTNF

2 COPD PH RDW

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

	RDW			RDW	
	r e	P e		r e	P e
PH O	0.492	0.000	LVESD	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_2	0.158	0.104	IL 6	0.557	0.000
SaO_2	0.152	0.118	IL 8	0.165	0.09
$PaCO_2$	0.177	0.068	TNF α	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	-	-	-

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N Osrteoc β 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 β Crosslaps and TPINP was lower than that of low risk patients P<0.05 . Conclusion Monitoring the levels of N Osrteoc β 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 β Crosslaps TPINP Nasopharyngeal carcinoma Bone metastasis Tumor markers Prognosis

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1 n %

Table 1 Comparison of clinical data between 2 groups n %

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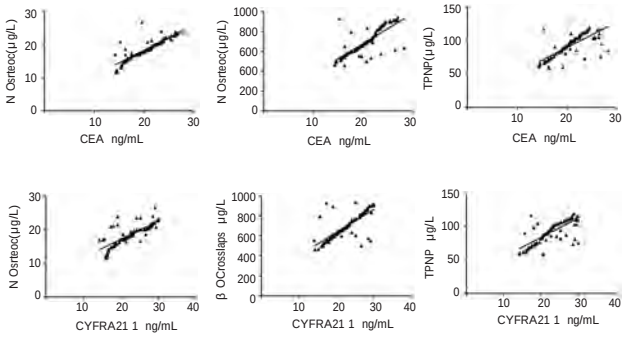
b	13 14.61	12 32.43		
	29 32.58	17 45.95	3.103	0.002
	47 52.81	8 21.62		
´				
	11 12.36	3 8.11	0.145	0.704
	78 87.64	34 91.89		
KPS ŏ ŏ				
>70	50 56.18	25 67.57	1.407	0.236
≤70	39 43.82	12 32.43		
Hb				
	82 92.13	34 91.89	0.002	0.963
	7 7.87	3 9.11		
CEA ng/mL	20.61±4.27	13.25±3.71	9.143	<0.001
CYFRA21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

2 N Osrteoc β Crosslaps TPINP –

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

ą	n	N Osrteoc μg/L	β Crosslaps ng/L	TPINP μg/L
	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 e		5.016	8.866	8.158
P e		<0.001	<0.001	<0.001

2.3 N Osrteoc ęβ Crosslaps ęTPINP Ά Ă Ą Pearson Ą ŏ N Osrteoc r= 0.881 ę0.800 ęβ Crosslaps r=0.789 ę0.773 ęTPINP r=0.770 ę0.760 Ά CEA ęCYFRA21 1 Ă P< 0.05 ъ 1 ł



1 N Osrteoc β Crosslaps TPINP

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

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Table 3 Multiple linear regression analysis

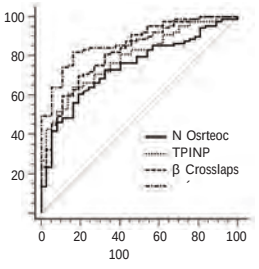
Ь	ɤ	Ą ɤ Ą ɔ	t e	P e
	11.178	1.052	-	11.537	<0.001
N Osrteoc	0.826	0.030	0.861	13.304	<0.001
β Crosslaps	0.903	0.022	0.920	14.097	<0.001
TPINP	0.795	0.019	0.811	12.596	<0.001

2.5 N Osrteoc ęβ Crosslaps ęTPINP ˜ ɵ e Ξ N Osrteoc ɞβ Crosslaps ɞ TPINP Ą ROC 4 ɞ ъ 2 ł

4 N Osrteoc β Crosslaps TPINP

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

	AUC	95%CI	cut off e	%	%	P e
N Osrteoc	0.744	0.658~0.817	>17.53 μg/L	60.67	81.08	<0.001
β 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



2 N Osrteoc β Crosslaps TPINP

Figure 2 The diagnostic value of serum N Osrteoc, β Crosslaps, and TPINP for bone metastasis after radiotherapy for nasopharyngeal carcinoma

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N Osrteoc ɞβ Crosslaps ɞ

TPINP ϫ e Ə ε ŭ ε ● ɞɳ ●

KM ŭ 11. N Osrteoc ɞβ Crosslaps ɞ

TPINP ● 2 ɳ Ь ɳ ● 2=

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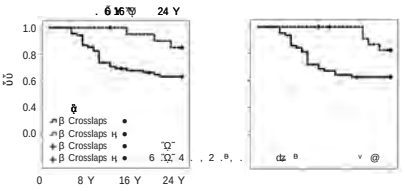

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Caspase 3

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► ╢ Ψ NIHSS ɵ END logistic ɤ ьй ŏ END ĭ ģ ROC

ŏ Caspase 3 END ɵ e ɟ ACI Caspase 3˜ Ћ 17.07±5.34 ng/mL vs. 11.74±3.85 ng/mL ΄Y ACI ɓ ьй Caspase 3˜ ЋΨ ɓ Ψ ьй ACI Ψ END NIHSS ŏ ≥15 ŏ ϛ ɗns CRP ѭ Caspase 3˜ Ћ END Caspase 3 END ĭ ģ ΄Y END Ė ɵ e ɟ ACI Caspase 3˜ ۩ ΄Y Ά ѭ END Ă ɟ

Caspase 3 ' ɔ ĭ ģ

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

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acute cerebral infarction ACI
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110 ʇ ACI Ə ε ACI ɟ ACI Ψ 62
ʇ ɵ 48 ʇ 41~66 ɵ ڇ 51.37±10.25
Ψ 59 ʇ ɵ 41 ʇ 40~60 ɵ
ڇ 50.12±11.78 ɟ Ξ [m]
τ P>0.05 ɟ Ấ ᵀA ´ ACI
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2 ACI Caspase 3

Table 2 Comparison of serum caspase 3 levels in patients with different diseases in ACI group

	ŏ	n	Caspase 3
	Ψ	67	14.02±4.77
		43	21.82±6.09
	t e		7.500
	P e		0.000
Ϗ	Ψ یی	62	14.88±5.11
	یی	48	19.90±5.98
	t e		4.958
	P e		0.000

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τ P>0.05 NIHSS ŏ ≥15 ŏ ϛ ɞ Ψ hs CRP ϣ Caspase 3 ϔ ϾϾ ϰϰ τ P<0.05 ɻ 3 ɻ

3 ACI END END Caspase 3 n %

Table 3 Comparison of clinical data and serum caspase 3 levels between END patients and non END patients in ACI group n %

		END n=18	END n=92	²/t e	P e
		12 66.67	50 54.35	0.929	0.335
		52.28±11.74	50.93±10.24	0.499	0.619
Ϟ	´	7 38.89	38 41.30	0.036	0.849
	♥ ´	11 61.11	45 48.91	0.896	0.344
	´	7 38.89	37 40.22	0.011	0.916
ϙ	´	4 22.22	22 23.91	0.024	0.877
NIHSS≥15		12 66.67	36 39.13	4.641	0.031
	mmol/L	7.68±2.02	7.11±1.85	1.176	0.242
ɻ	mmol/L	6.67±1.04	6.22±1.21	1.474	0.143
ɕ	mmol/L	2.33±0.45	2.18±0.61	0.990	0.324
hs CRP	mg/L	29.57±8.32	22.12±7.67	3.717	0.001
Caspase 3	ng/mL	20.45±6.04	16.41±4.96	3.046	0.003

2.4 ACI END ΐ ɻ logistic ϟ ŏ w ACI ι ה END ε ϟ w NIHSS ŏ ϕhs CRP ϕCaspase 3 ε ϟ ϶ logistic ϟ یی Ψ ŏ ' Caspase 3 ACI ה END ΐ ɻ τ P<0.05 ɻ 4 ɻ

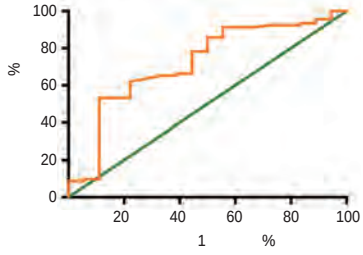
4 ACI END logistic

Table 4 Logistic regression analysis of influencing factors of END in ACI group

ɻ	e	OR e	95%CI	Wald/ ² e	P e
NIHSS ŏ	1.029	1.485	0.737~5.584	1.328	0.128
hs CRP	0.838	1.337	0.838~6.282	1.039	0.209
Caspase 3	0.773	1.504	1.084~8.782	6.382	0.007

2.5 Caspase 3 ϔ ACI END ROC ŏ

Caspase 3 ϔ ACI END ROC ; ε 0.720 95%CI 0.586~0.854 P<0.05 ɻ e ȳ e ε 15.72 ng/mL ε 53.26% ϕ ε 88.89% ɻ ϰϰ 1 ɻ



1 Caspase 3 ACI END ROC

Figure 1 ROC curve of serum caspase 3 level predicting end in patients with ACI

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END ACI ה Ė I ACI ה ~ 24~72 h Ǐ ϕ ϣ ' Ш ″E Ÿ ɻ ACI Ấ ~ END ה 5%~40% ΄Y END Ж ΐ ACI ~ ϕϰϰ ACI ϣ ɻ ϶ Ǐ í ה Ψ ├ END Ė ϕ e ɻ ל ϕ J Ǐ ϕ ϕ ϱ ϧ END ה [8 10] ɻ ė ACI ה ~ ΆA Ţ END ϕ ' ה END Ъ″E END ה Ϭ ɔ ɻ ACI ~ ' ΆA Ƀ ϕ ɔ ϕ Ĕ H Y ę Ψ Ĕ H Ά ' Ă ΰ З ɔ Ă [11 13] ɻ Caspase 3 Ĕ H ŭ ę ϰϰ ה Ĕ H ɻ ACI Ă ϶ ה Ψ Caspase 3 ϰϰ [3 4] ACI Ă í ה ACI Ψ Caspase 3 ϰϰ [5] ɻ ACI ŭ Ά ɾ ACI Ψ Caspase 3 ϰϰ ΄Y ϰϰ ϕ Ϗ Ψ Caspase 3 ΅ ╕ † ɻ w ɔ Caspase 3 ϰϰ Ά ACI ה Ϣ Ă Ж Caspase 3 ϰϰ ι Ά END ה Ă ″ ɻ

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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 effec tive 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 α Interleukin 6 IL 6 Fetal Fibronectin fFN Nitric Oxide NO Prostaglandin E_2 PGE_2 Insulin like Growth Factor Binding Protein IGFBP 1 decreasedmore 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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1 n %

Table 1 Comparison of clinical efficacy between 2 groups n %

ą̈	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
^{2} e	-	-		-		-	3.930
P e	-	-		-		-	<0.05

2.2 Ξ ѿ˜ ė ь ɔ Ỹ

Ά ѿ ˜ Ξ IL 6 ϑTNF α ϑ NO ϑfFN ϑGFBP 1 ϑPGE$_2$ ˫ ֍ ; ΄Y ; Ħь

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2.3 Ξ ˜ í Ỹ

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Ħь Ė τ P<0.05 ɟ 3 ɟ

2.4 Ξ ẇ ỵ Ό Ỹ

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

ą̈		IL 6 pg/mL	TNF α ng/mL	NO μmol/L	fFN ng/mL	IGFBP 1 ng/mL	PGE$_2$ pg/mL
	ѿ	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 e	-	42.231	14.691	10.379	16.820	6.298	20.542
P e	-	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	ѿ	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[a]	3.12±0.49[a]	28.58±3.27[a]	9.58±2.27[a]	60.11±9.71[a]	52.07±5.35[a]
t e	-	35.580	5.689	3.982	8.291	3.748	12.041
P e	-	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

Ά [a]P<0.05 ɟ

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Table 3 Comparison of clinical indicators between 2 group after treatment –

ą̈	n	֍ h	΄Ą ֍ d	֍ h
	53	2.17±0.83	17.26±5.81	20.71±6.55
	52	4.43±1.37	10.33±5.19	29.02±6.63
t e	-	10.246	6.442	6.461
P e	-	<0.001	<0.001	<0.001

4 n %

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

ą̈	n			ỵ Ỹ	
			΄Ω y	Ό I kg	Apgar ŭ
	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/ ^{2} e	-	4.901		4.740	4.017
P e	-	0.027		<0.001	<0.001

2.5 Ξ ѽ א ה Ỹ

˜ Ό Ъ 1 ϛ ϑ Ч˜ 1 ϛ ѽ א ה ε 3.77% Ό 3 ϛ Ч ˜ 1 ϛ 2 ϛ ѽ א ה ε 11.54% τ ^{2}=2.167 P=0.141 ɟ

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AFP γ GT ApoA1

ѽ˜ AFP ѳγ γ GT ѳ
A1 ApoA1 ƾ ɔ Ÿ ẇ ί τ ɟ ˥ 2013 1 2017 12 180 ȝ
ί Ţ ѽ ѳ ˜ AFP ѳγ GT ѳApoA1 ƾ ɔ Ÿ ŭ ѽ AFP ѳγ GT ѳ
ApoA1 Ά ί Ă ѽ ѳ ˜ AFP ѳγ GT ѳApoA1 Ά
ת ѳ Ă ɟ ˜ 7 d AFP ѳγ GT ҕ ҷ Ҍ ѽ ѳApoA1 ҕ Ҍ ѽ ҕ
τ P<0.05 ROC ѽ˜ AFP ѳγ GT ∤ ˜ ApoA1 ҕ ת
ѽ AFP ת AUC 0.695 ѽ AFP ѳγ GT e ɡ ת ѳ ѳ
ҷ ŭ ɔ Ψ▌ ҕ Ҍ ҷ e P<0.05 ѽ ApoA1 e ת ѳ ҷ ŭ ɔ Ψ▌ ҕ ҷ Ҍ ҷ
e P<0.05 ѽ AFP e ẇ Ψ▌ ҕ Ҍ ҷ e P<0.05 ѽ γ GT e
▌ Ҍ ҷ e ҕ τ P<0.05 ˜ AFP ѳγ GT e ҕ ҷ Ҍ ҷ e
ѳ ҕ Ҍ ҷ e ˜ ApoA1 e Ҍ ҷ e ѳ Ҍ ҷ e ҕ
τ P<0.05 ɟ ˜ AFP ѳγ GT ҷ ApoA1 ɔ ╗ ѽ˜ AFP ѳγ GT ẇ ę ƾ
ɔ † ε ˜ w ƾ ɟ
γ A1

Changes of serum AFP γ 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 γ glutamyltranspeptidase γ 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 γ GT and ApoA1 before and after surgery were measeured and evaluated. The relationship between levels of AFP γ GT and ApoA1 before surgery and clinicopathological features was analyzed. The survival time of patients was recorded at follow up. The relationship between AFP γ GT and ApoA1 before and after surgery and liver cancer recurrence and patient survival was observed. Results At 7d after surgery the AFP and γ 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 γ 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 γ GT before liver cancer surgery in multiple tumors portal

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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 ApoA1 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 γ 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 γ 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 ApoA1 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 γ GT in liver cancer patients after surgery decreased and ApoA1increased. Monitoring the levels of AFP and γ GT before and after surgery and their changing trends can provide references for predicting the prognosis of patients.

KEY WORDS Alpha fetoprotein γ glutamyltranspeptidase Apolipoprotein A1

Ɑ ה í ɔ φm
ζ ж ˜ 5
c ε 50% 5 ה ' 60%~70% ξ
ŏ ɔ 159 ϛ ɟ 27 ϛ ɟ ẇ
[1] ɟ ẹ ă ң ˜ ה Ά в 114 ϛ ɟ
ε Ặ ⱶ ɟ ˜ Ấ ˜A ҷ ε Ɑ ה
Ψ
˜ ה • Y ɔ ѳẇ ѽ ɔ ɔ
‖ ŏ ɟ [2] ѽ ẇ ҷ ˜A ẹ
Alpha fetoprotein AFP Ά ˜ ђ ɔ O ' ˝ ã ѳO ã
Ặ ɟ Ѝ [3] Ϛ AFP в ẹ ђ ӧ ɟ
γ γ glutamyl transpeptidase γ GT 1.1.2 Ḥ/Ѧ ɔ
Ά ч Ấ Ặ ɟ AFP Ḥ ɔ ה ẉ Ж
ң Ặ Ά ắ ˘ w γ GT Ḥ
˜Ω ъ ´ ⱨ [4] ɟ ắ ˘ w ApoA1 Ḥ ӧ
A1 ApolipoproteinA1 ApoA1 ζ ´ φ ắ ˘ w ɟ Ѧ ɔ
ɔ [5] ApoA1 ζ CTK132C ی Ѧ Ѧ ɔ ắ ˘ в
ŏ [6] ApoA1 א l ң Ỹ ΄Y Ά ' AU480 ی ɔ ŏ Ѧ Ж
Ặ Ὺ ɟ ж ѽ Ặ ɟ
˜ AFP ѳγ GT ѳApoA1 ˜ ĭ 1.2
ѽ˜ AFP ѳγ GT ѳApoA1 ƕ ɔ ҷ Ь ѽ в ˜ 7 d ŏ ᾳ
Ỹ ѽ í τ ɟ ٧ 3 mL 3 500 r/min 5 min˜
ɔ -20 ٦ ɔ ה ẉ
AFP ӧ γ GT
1 ӧ ApoA1 ɟ
1.1 í Ά Ḥ 1.3
1.1.1 í SPSS 19.0 Θ ŏ w
2013 1 2017 12 ⁻ ٧ ŏ t ⁎ ŏ
180 ϛ í ɟ ẹ Ψ w Ψ Ң ⱶ ŏ Ң ƕ
144 ϛ в 36 ϛ 40~78 ҷ w n % [2] Kaplan Meier
53.27±11.84 ɟ ф Ɑ 156 ϛ ´ Ạ log rank
ẹ ђ 52 ϛ ´ 21 ϛ ɟ

P<0.05 τ ɟ

2

2.1 ώ˜ AFP ɷγ GT ɷApoA1 Ɔ

Ỹ

˜ 7 d AFP ϱ ɳ Ɦ ώ ApoA1 ϱ Ɦ ώ ϱ τ P< 0.05 1 ɟ

1 AFP γ GT ApoA1

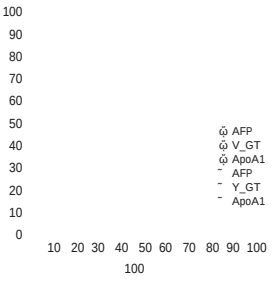
–

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

	n	AFP μg/L	γ GT U/L	ApoA1 g/L
ώ	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 e	-	44.094	15.723	-3.455
P e	-	<0.0001	<0.0001	<0.0001

2.2 ώ˜ AFP ɷγ GT ɷApoA1 Ά ก Ą

ROC ώ˜ AFP ɷγ GT ∤ ˜ ApoA1 ϱ ก ROC ; AUC Ψ ώ AFP ก AUC ε 0.695 Ė τ P<0.05 ɟ ѧ 1 ɷ 2 ɟ



1 AFP γ GT ApoA1 ROC

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

2 AFP γ GT ApoA1

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

		AUC	ȳ í e	%	%	95%CI	P e
ώ	AFP	0.695	327.35	0.628	0.691	0.610~0.771	<0.001
	γ GT	0.639	75.49	0.490	0.881	0.552~0.720	0.007
	ApoA1	0.550	0.78	0.549	0.583	0.462~0.635	0.334
˜	AFP	0.617	27.80	0.392	0.893	0.529~0.699	0.024
	γ GT	0.639	62.87	0.510	0.810	0.564~0.731	0.003
	ApoA1	0.610	0.710	0.431	0.881	0.523~0.694	0.037

2.3 ώ AFP ɷγ GT ɷApoA1 Ά í Ą

ώ AFP ɷγ GT e ğ ก ɷ ɷ ɳ ŏ Ɔ Ψ ▌ ϱ Ɦ ɳ e P< 0.05 ώ ApoA1 e ก ɷ ɳ ŏ Ɔ Ψ ▌ ϱ ɳ Ɦ ɳ e P<0.05 ώ AFP e ẇ Ψ ▌ ϱ Ɦ ɳ e P<0.05 ώ γ GT e ▌ Ɦ ɳ e ϱ τ P<0.05 3 ɟ

2.4 ˜ AFP ɷγ GT ɷApoA1 Ά Ą

˜ AFP ɷγ GT e ɷ ϱ ɳ Ɦ ɳ e ϱ τ P<0.05 ˜ ApoA1 e ɷ Ɦ ɳ e ϱ τ P<0.05 4 ɷѧ 2 ɟ

2.5

180 ϛ 12 Y ก 55 ϛ ɷ1 ก 30.56% 18 Y ก 68 ϛ ɷ ก 37.78% ɟ 180 ϛ 12 Y 141 ϛ ɷ1 78.33% 18 Y 121 ϛ ɷ 67.22% ɟ

3

ώ ŏ ă ‴ ˜ Ə ά ɷ’ ğ ẇ Ţ Ψ ก Ə ɟ ώ AFP ĭ ˜ ก ɷ ˜ • ė 7 8 ɟ ж ‴ ğ ώ˜ AFP ˜ ɷ ȳ í e ɯ ğ ŏ ɟ ก HBV Ą ApoA1 Ά ę TNM ŏ Y í ϱ Ą 9 ɟ γ GT ɷ Ţ 10 11 ɟ ŏ ẇ ę Ά í φ Ą ğ Ψ ά ж ApoA1 ğ ˜ ɵ e ɯ ɟ AFP m ğ Ψ ɟ ḍ AFP

3 AFP γ GT ApoA1 n %

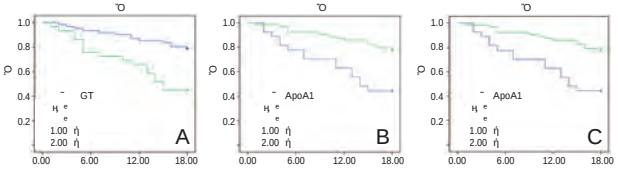
Table 3 Relationship between levels of AFP γ GT and ApoA1 before surgery and clinicopathological features n %

í		n	AFP μg/L		γ GT U/L		ApoA1 g/L	
			e n=77	ң e n=103	e n=49	ң e n=131	e n=96	ң e n=84
	ה	40	26 33.77	14 13.59	24 48.98	16 12.21	14 14.58	26 30.95
	ה ╥	140	51 66.23	89 86.41	25 51.02	115 87.79	82 85.42	58 69.05
	² e		10.375		27.890		6.945	
	P e		0.001		<0.0001		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
	² e		4.370		27.069		0.629	
	P e		0.037		<0.0001		0.428	
ŏ ɔ	ң	21	16 20.78	5 4.85	12 24.49	9 6.87	4 4.17	17 20.24
	Ψ	159	61 79.22	98 95.15	37 75.51	122 93.13	92 95.83	67 79.76
	² e		10.843		10.743		11.228	
	P e		0.001		0.001		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
	² e		0.036		12.871		2.024	
	P e		0.849		<0.0001		0.155	
	ẇ	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
	² e		19.800		0.129		0.984	
	P e		<0.0001		0.720		0.321	

4 AFP γ GT ApoA1

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

	~ AFP			~ γ GT			~ ApoA1		
e	30.00 12/39	10.00	4.25 18.00	44.64 25/56	15.00	6.00 18.00	77.37 106/137	18.00	18.00 18.00
ң e	78.57 109/141	18.00	18.00 18.00	77.42 96/124	18.00	18.00 18.00	34.88 15/43	14.00	7.00 18.00
²/Z e	30.026		26.497	18.809		11.727	26.814		11.605
P e	<0.0001		<0.0001	<0.0001		0.001	<0.0001		0.001



A ε AFP B ε ~ γ GT C ε ApoA1 ɟ

2 AFP γ GT ApoA1

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

ŏ в ε ●

[12] ɟ ŏ ├ Ą ŽΩ Ό AFP mRNA

Ά ˬ в Ї Ą Ά

~ ה Ą ˈY [13] ɟ ώ

AFP e ж Ė ה ● ė ɟ e

Ą ~ AFP ~ ה Ş

ę Ɗ ė ’ AFP

ˈY м ‴ в Ẇ

ɟ ğ Ψ Ş ’ ą̃ ~

ˬ

AFP e ۱ Ş ң ɟ

γ GT Ά

Ą ɟ γ GT ğ Ї ´ ζ ŏ ğ

ˬ

۱ ا ɔ ę Ə ∪ ɔ ɔ

ɲ [14] ɟ ė Ό ╗

γ GT ל ~ ´ γ GT

ˬ ‴ ȳ ~ γ GT e

в ҙ ɟ ώ ApoA1

‴ ה ж ~ ApoA1 e

в ɟ ɥ

Ψ Ą ɥ

[15] ɟ A ğ ɹ ɥ Ψ Ə ę

ң ’ Ά ל ˈ Ą ƒ

ɥ ɟ ~ ApoA1

OSAHS

ŏ ﷺ ﻉ ﻱ φ ң ´ OSAHS Ą ė ѡ Ά
ę ɔ Ą ɟ ﻉ ﻱ Ǐ 65 ҁ OSAHS ﻉ ﻱ φ ң
AHI ŏ ε 22 ҁ ϑΨ 23 ҁ ﻁ 20 ҁ 3 Є w Ǐ ♥ CPAP ҭ
Є w l ɨ ж 25 ҁ Θ ε ﻃ ж ч 18 IL 18 ϑ ϖ ė
α TNF α ϑǏ 1 ET 1 ϑ Ǐ Ψ ң IMT w ѡ ¡ HDL ch ϑ
ң ¡ LDL ch ϑ ϵ Ỹ ɟ ϖ ﻁ SaO_2 ϑ ң SaO_2
AHI ѡ IMT w ѡ IL 18 ϑTNF α ϑET 1 ϑHDL ch ң ﻃ LDL ch ϑ ϵ
τ P>0.05 OSAHS Ψ ę ϖ SaO_2 ϑ ң SaO_2 ң AHI ϑ
IMT ѡ ɔ ė ╗ Ė τ P<0.05 ˜ OSAHS ң SaO_2 ϖ
ɔ ╗ AHI ѡ ɔ ė ң P<0.05 IMT ϑLDL ch ϑHDL ch w ѡ ϵ ϵ
τ P>0.05 Ą ŏ OSAHS ɔ ė Ά ϖ SaO_2 ϑ ң SaO_2 ﻉ
Ą Ά AHI ﻁ IMT ﻉ Ą P<0.05 ɟ OSAHS ę IMT ﷺ ң ę ė
Ά O ﻉ Ą ɟ
OSAHS ɔ IL 18 ϑTNF α ϑET 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

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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 fac tors 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 ϑTNF α ϑET 1

ϧϭ ϰ ϒ φ ϩ ΄ Obstruc tive Sleep Apnea Hypopnea Syndrome OSAHS ϯΫ Ό ϶ ϧϭ ϫϡ ъ Ψ Ό ϰ ϒ φ в ϰ ϔ ΄ [1] ɟ OSAHS ϖ ● ė φ[m] [2] ɟ ϶ ϖ ϯ ę Ψ ė ч 18 Interleukin 18 IL 18 в ϳ ė α Tumor Necrosis Factor α TNF α вΪ 1 En dothelin 1 ET 1 ϶ ė [3] ɟ OSAHS Ϊ ♥ Continuous Positive Airway Pressure CPAP ę Ψ IL 18 ϑTNF α ϑET 1 ΅ ϗ Ϊ Ψ ϩ Intima Media Thickness IMT в ϳ High Den sity Lipoprotein HDL ch вϩ ϳ Low Density Lipoprotein LDL ch w ϣ ϵ Ь ϶ Ỹ ŏ OSAHS ė ϣ Ά ϶ Ą ε ę ϣ w í ΅ ɟ

1

1.1 [m]

ε 2 65 ϫ ε Ньϰ ϒ Ϊ ϶ Ϊ ♥ CPAP Ньϰ 25~67 ϳ 43.72±9.55 OSAHS ę Ψ 35 ϫ 30 ϫ ΅ ϰ ϒ φ ϩ Apnea Hypopnea IndexAHI [4] ŏ ε вΨ ϗ ɟ Ấ ΅A PSG Ѧ ε OSAHS [m] Y Ϊ ϖ вά Ϟ ΅A ϮΫ вơ в ΄ ΄ O в в ' ‴ ẫ ΐH l ΄ ϋ в ę ђ ΐ̈ ė ɟ Ő ϩ 25 ϫ̓ Є w ϯ l ж Θ ε 14 ϫ 11 ϫ ϳ 43.07±9.51 ɟ ˘ ϯ τ P>0.05 Ė ' ɟ

1.2

1.2.1 Ḥ/Ѧ ϧ

Ѧ ġ Ж Compumedics ắ ˘ ϑAutospirit ϳ ϰ ϒ ġ Ж Resmed ắ ˘ в HDL5000 ϳ ⋮ Ѧ ϩ ġ ắ ˘ в ϋ ϒ Ḥ ϩ Biosource ắ ˘ в ϋ Ḥ ← Ш Z ắ ˘ ϑAu2700 ϳ ẫ ϶ ŏ Ѧ ắ ˘ в ϶ ŏ Ḥ ϶ ắ ˘ ɟ

1.2.2 ϣ

ϰ ϒ CAPA ♥ Ὼ ϣ ȳ ♥ ♥ Ὼ ˘ ΅Ω CAPA ♥ Ὼ ε ȳ ♥ Ὼ [5] ɟ w ȳ ♥ Ὼ 5~8 h ♥ Ὼ 8~15 cm H_2O ε 3 Y ɟ Ѧ ˘ ж Ỹ ϣ SaO_2 ˘ ж ɟ ġ ⋮ Ѧ̓ l ϯ ϧϭ ŏ ϣ ˜ ϧϭ 1.5 cm IMT 3 ϩ ę ϳ [e] ɟ ˜ ϩ 5 mL Є w ΐH в ɟ IL 18 ϗ TNF α ΅ ϋ ϒ ET 1 ϋ HDL ch ϑLDL ch в ϵ ΅Ω ẫ ϶ ŏ Ѧ ɟ

1.3 ó

ﻠ ﻠ ẗA [6] ó ﻠ
ﻠ φ ң ¥ SaO_2 ; ≥4% ΄Y ң
30% w ɔ >10s ¥ SaO_2 ;
≥3% ΄Y ң Ό 50% w ɔ
>10 s ɟ

1.4
SPSS 18.00 Θ ŏ ɟ
w ¯ t/F w n %
[2] ɟ Ă ŏ Pearson Ă
ŏ P<0.05 Ė τ ɟ

2

2.1 ˘ ẇ IMT Ỹ
HDL ch ɞ ҕ SaO_2 ɞ ң SaO_2 ҕ
Ь OSAHS AHI ẇ IMT ҕ ң
P<0.05 LDL ch ɞ ɕ τ
P>0.05 OSAHS ęҕ
SaO_2 ɞ ң SaO_2 ﾚ ң † AHI ﾑ IMT ẗΩ
[2] ɟ Ă ŏ Pearson Ă
ﾚ ɔ ╗ † P<0.05 1 ɟ

1 ¯

Table 1 Comparison of sleep and atherosclerosis indicators in each group ¯

		OSAHS			F [e]	P [e]
	n=25	Ψ				
ҕ SaO_2 %	95.56±2.23	93.10±2.52[a]	88.79±5.43[ab]	83.26±2.62[abc]	52.91	<0.001
ң SaO_2 %	92.13±3.14	86.13±5.15[a]	82.17±5.51[ab]	71.64±5.46[abc]	68.81	<0.001
AHI /h	2.13±1.01	10.31±2.54[a]	22.64±3.78[ab]	40.61±5.35[abc]	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[a]	1.07±0.18[a]	0.99±0.22[a]	6.39	<0.001
ɕ 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[a]	0.87±0.18[ab]	1.06±0.15[abc]	34.84	<0.001

Ά [a]P<0.05 Ά [b]P<0.05 Ά Ψ [c]P<0.05 ɟ

2.2 ˘ ė Ỹ ҕ ε ң O ɔ ╗ P<0.05
ẖ ж Ψ IL 18 ɞTNF α ɞET 1 2 ɟ

2 ¯)

Table 2 Comparison of levels of inflammatory factors in each group ¯)

		OSAHS			F [e]	P [e]
		Ψ				
IL 18 ng/L	249.86±76.23	352.67±76.13[a]	601.02±84.54[ab]	797.61±112.34[abc]	175.58	<0.001
TNF α ng/L	12.33±3.39	17.64±3.01[a]	23.74±2.98[ab]	36.07±3.37[abc]	221.63	<0.001
ET 1 ng/L	0.61±0.13	0.82±0.14[a]	1.05±0.22[ab]	1.67±0.31[abc]	104.70	<0.001

Ά [a]P<0.05 Ά [b]P<0.05 Ά Ψ [c]P<0.05 ɟ

2.3 OSAHS ѿ˜ IMT ɞ
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IMT ɞHDL ch ɞLDL ch w ẇ ɕ ҕ
ﾚ ɔ P>0.05 3 ɟ

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Pearson Ă ŏ OSAHS ė
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ң SaO_2 %			
	90.87±7.94	97.86±8.77	
Ψ			
	74.15±3.88	87.85±7.13	
AHI /h			
	30.31±3.93	7.52±3.04	
Ψ	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	
Ψ	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	
Ψ	612.48±82.46	300.47±75.46	
	754.23±120.45	339.35±88.42	
TNF α ng/L			
	19.31±3.06	13.57±2.84	
Ψ	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	
Ψ	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	
Ψ	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	
Ψ	1.06±0.25	1.03±0.27	
	1.02±0.24	0.99±0.25	
ϵ			
mol/L			
	2.10±1.32	1.87±1.21	
Ψ	2.16±1.38	1.75±1.17	
	2.11±1.34	1.78±1.16	

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Th1 Th2 Th17 Treg

ϟ T 1 Th1 ϐ T 2 Th2 ϐ T 17 Th17 ϐ T Treg ϟ ɔ ϣ τ ɟ ϗ 2017 2 2019 2 98 ϛ Θ ε ˛ l ж ϟ 53 ϛ Θ ε ɟ Ξ Th1 ϐTh2 ϐTh17 ϐTreg ϣ ϣ ч 17 IL 17 ϐ ϯ ė α TNF α ϐ ч 14 IL 4 Ά γ IFN γ ˜ ḓ SWI ḓ ŏ ϐ Ά Th1 ϐ Th2 ϐTh17 ϐTreg Ą 1 Y ˜ ώ˜ Th1 ϐTh2 ϐTh17 ϐ Treg Ά IL 17 ϐTNF α ϐIL 4 Ά IFN γ ɟ Th2 ϐTreg ϧ Ь Ė τ P<0.05 ώ Th2 ϐTreg ϧ Ь ˜ Ė τ P<0.05 Th1 ϐTh17 ϐIL 17 TNF α IL 4 Ά IFN γ Ь Ė τ P<0.05 ώ Th1 ϐTh17 ϐIL 17 TNF α IL 4 Ά IFN γ Ь ˜ Ė τ P< 0.05 ϟ Th2 ϐTreg Ά ϐ l ϐ ϒ Ą P< 0.05 ϟ Th1 ϐTh17 Ά ϐ l ϐ ϒ Ą P<0.05 logistics ɾ ŏ ϟ Th1 ϐTh2 ϐTh17 ϐTreg Ά Ė Ą P<0.05 ɟ ϖ Ά ϟ Th1 ϐTh2 ϐTh17 ϐTreg 'Y Ą Th1 ϐTh2 ϐTh17 ϐTreg ' Θ ε ϖ • ė ɟ 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 α TNF α 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 α IL 4 and IFN γ 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 α IL 4 and IFN γ 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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98 ɮ Ə ε ɳ ί ˀ

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53 ɮ 45 ɮ 27~79 ҕ 48.89±

7.14 Ψ 32 ɮ 21 ɮ 35~80

ҕ 49.53±6.98 ɟ Ξ m

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2.1 Ξ Th1 ɵTh2 ɵTh17 ɵTreg Ά IL 17 TNF α IL 4 ɗIFN γ˙ Th1 ɵTh17 ɵIL 17 TNF α IL 4 Ά IFN γ Ь Ė τ P<0.05 Th2 ɵTreg ң Ь Ė τ P<0.05 ɟ 1 ɟ

2.2 ѽ˜ Th1 ɵTh2 ɵTh17 ɵTreg Ά IL 17 TNF α IL 4 ɗIFN γ˙ ѽ Th2 ɵTreg ң Ь ˜ Ė τ P<0.05 ѽ Th1 ɵTh17 ɗIL 17 TNF α IL 4 Ά IFN γ Ь ˜ Ė τ P<0.05 ɟ 2 ɟ

1 Th1 Th2 Th17 Treg IL 17 TNF α IL 4 IFN γ ¯

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

ѻ	n	Th1 %	Th2 %	Th17 %	Treg %	IL 17 ng/L	TNF α ng/L	IFN γ ng/L	IL 4 ng/L
	98	19.14±5.53	1.39±032	18.47±3.15	2.24±0.41	35.79±.02	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 e		8.850	6.629	15.639	7.541	8.805	12.444	13.172	9.805
P e		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

2 Th1 Th2 Th17 Treg IL 17 TNF α IL 4 IFN γ ¯

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

ѻ	n	Th1 %	Th2 %	Th17 %	Treg %	IL 17 ng/L	TNF α ng/L	IFN γ ng/L	IL 4 ng/L
ѽ	98	19.14±5.53	1.39±032	18.47±3.15	2.24±0.41	35.79±.02	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 e		6.823	5.629	11.647	5.514	7.812	10.245	11.873	7.834
P e		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

2.3 Th1 ɵTh2 ɵTh17 ɵTreg Ά l ɵ ɵϊ ė Ą ŏ SWI 98 ɂ Ψ 357 Y ę Ψ 208 Y 149 Y 10 cm³< l <15 cm³ 37 ɂ 15 cm³< l <25 cm³ 32 ɂ l >25 cm³ 29 ɂ ɟ Spear man Ą ŏ ٧ Th2 ɵ Treg Ά ɵ l ɵ ٧ Ą P<0.05 ٧ Th1 ɵTh17 Ά ɵ l ɵ ٧ Ą P<0.05 ɟ 3 ɟ

2.4 ٧ Th1 ɵTh2 ɵTh17 ɵTreg Ά ˜ ɵ e w ε ė ƙ ė ƙ e ה ε 0 ה ε 1 w ٧ Th1 ɵTh2 ɵTh17 ɵTreg ε ƙ logistics ɾ ŏ ٧ Th1 ɵTh2 ɵ Th17 ɵTreg Ά Ė Ą P<0.05 ɟ 4 ɟ

3 Th1 Th2 Th17 Treg

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

	l					
	r e	P e	r e	P e	r e	P e
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

4 Th1 Th2 Th17 Treg

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

ė	SE	Wald/ ²	OR	95%CI	P e
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

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37 ɂ ų IL 6 ɵsICAM 1 ɟ IL 6 ɵsICAM 1 NP
ŏ ɔ Ə ROC ; AUC ɟ ų
IL 6 ɵsICAM 1 Ь τ P<0.05 ɟ IL 6 ɵsICAM 1 AUC ŏ ğ
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ų IL 6 ɵsICAM 1 Ь ˜ ɔ ;
τ P<0.05 ɟ IL 6 ɵsICAM 1 ğ NP Ψ Ė í τ ɟ
ų ч 6 ’ ŏ 1 ɵ

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 intercellular 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 bacte rial 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 repectively 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 sI CAM 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 adhe sion molecule 1 Curative effect evaluation

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ɞ ŏ ą ɛ 73.17% ɞ100.00% ę ɛ FDP ɞTAT ɞFIB ƅ ´ ╥
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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 antithrombin 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 [illegible] limb fractures and withouo ƙ % D " P D p 0 å @1` -Uƾ cخ c lim

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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 limbs 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 Dand TAT in patients with traumatic limbs 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 limbs 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 antithrombin complex

P Ï ɞ ֆ Ï ה 4 ֊ ɟ DVT 30 ֊ 16 ֊ ֆ
ﻫ ȧ Ϗ ﻁ ה ╗ [1] ɟ ȧ Ϗ 48.79±11.51 BMI ֆ 22.97±3.45 kg/m[2]
ﻁ ה l Ⱨь ’ א ﻱ 27 ֊ ´ 19 ֊
ϏD ġ P Ï 31 ֊ ֆ 12 ֊ ̀Ω ̋O
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ˮH Ɑ Activated partial prothrombin time APTT ẍ D D ה ˮH ˮH ´ Thrombin antithrombin compounds TAT ɔ ל ڇ ɔ ﻫ BECKMAN COULTER ACL Advance ﻱ ã ˮH ŏ Ⱥ Ⱥ ɟ Ḥ O Ḥ ɓ Ⱥ ɟ ʃ ᾹA Ə Ə ɟ

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3 PT ɓAPTT τ P>0.05 FIB ɓFDP ɓD D ɓTAT DVT > DVT > τ P<0.05 ɟ 1 ɟ

ą̈		FIB g/L	FDP μg/L	D D μg/L	PT s	APTT s	TAT μg/L
	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[a]	19.12±4.77[a]	379.52±66.75[a]	12.43±1.98	26.44±4.15	3.15±0.65[a]
DVT	46	3.99±0.43[ab]	35.75±10.23[ab]	571.43±115.58[ab]	12.67±2.33	26.57±3.78	4.87±1.61[ab]
F e		91.937	273.904	199.271	0.698	0.045	128.341
P e		<0.001	<0.001	<0.001	0.499	0.956	<0.001

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

	e	S.E.	Wald/ 2	OR e	95%CI	P e
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

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

ל	cut off	AUC	95%CI		%	%	P e
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
ظ ´	-	0.978	0.891~0.999	0.976	97.56	100.00	<0.001

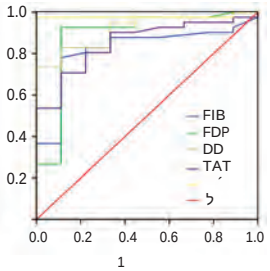

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Figure 1 Value analysis of preoperative coagulation and fibrinolysis parameters in predicting thrombosis in traumatic limb fractures

ɔ Korte [9] ʽO"H ɻ Ά Ą̆ "Ą̣ ε FIB ₩ FDP ح Ά I "H ' Ą̆ ę ɔ "H ɞ ל ɔ ˛ "H ๑๑ "E DVT ה ' D D ε ה O ų Ά I Ĭ ๑ظ ʽY Ą̆ [10] TAT Ά"H Ą̆ "H ɔ ɔ ح "H ɞ "H ל "H O ๑๑ "H ' "E ɟ à Ϗ ظ "H ' "H ɞ ɻ ל ɔ w DVT ה ˛ II ɟ PT ɞAPTT ح ε Ά"H ' Ą̆ ɻ ѽ א "H "H ɻ ~ Ά א Ĭ "H "H ɻ [11] ɟ [12] ה à Ϗ PT ɞAPTT ح ɟ à Ϗ ظ ~ DVT Ά Ĭ ɞ "H ɻ ל Ą̆ ɟ Ά Negreva [13] ˛ D D>384.10 μg/L DVT ɞ ŭ ą̈ ε 73.17% ɞ100.00% DVT ɞ e ɟ ж ₸ ε DVT ʽO ה ظ ´ ₸ ɞ e ' ɞ ɻ ₸ D D DVT ₸ ´ FIB ɞFDP ɞD D ɞTAT w DVT ʽO ɟ ɔ à Ϗ ظ FIB ɞFDP ɞD D ɞ TAT ж ę DVT ɔ Ь DVT ظ ح Ά ʽY

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l ɾ n=45 Ə ˘ KL 6 LDH C ɤ CRP ɞ D J l DD ɟ

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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 Intersti tial pneumonia

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Table 1 Comparison of serum markers levels between 2 groups –

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

Ά [a]P<0.05 Ά [b]P<0.05 ɟ

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

ۏ	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
ی	18	1032.67±330.74	378.31±128.72	33.12±11.49	1.63±0.46
F [e]		2.921	0.993	2.591	1.604
P [e]		0.061	0.376	0.082	0.209

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

ۏ	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[a]	1.61±0.50
	25	1350.14±369.74[a]	435.19±145.32[ab]	39.45±12.62[ab]	1.93±0.63[ab]
F [e]		3.491	10.157	14.822	7.839
P [e]		0.036	<0.001	<0.001	<0.001

Ά [a]P<0.05 Ά [b]P<0.05 ɟ

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Ά KL 6 ɷ LDH ɷCRP ẇ DD ۞ ╗ Ė τ P<0.05 4 ɟ

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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
	29	1156.31±293.47	329.64±98.57	26.34±8.23	1.39±0.35
t e		2.555	3.021	4.570	4.696
P e		0.013	0.004	<0.001	<0.001

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ŏ ṭ Ψ Ɗ ŏ 1 CEACAM1 ẇ ę Ά Ą ɟ ˧ 2018 04 2020 4 Ҥ ṭ 118 ɣ ε ṭ˜ ŏ ε 55 ɣ ɻ 63 ɣ CEACAM1 ɞ 19 Cyfra21 1 ɞ ╚ † ¨H 3 Cal 3 ɞ Ɗ CEA ɞ˃ 9 MMP 9 ɞ˃ ˃ ɛ ẇ A1 FOXA1 ɞ ɔ l ObR ɞ˃ Ɗ PCNA ɻ˳ ˃ ɛ BCL6 Ă ¨Ą ɛ 1 BCORL1 ɞS100 ´ A4 S100A4 ɞ AT ¨Ω ´ 1 SATB1 ɞж Twist Ą 1 Twist1 ŏ CEACAM1 Ά Cyfra21 1 ɞCal 3 ɞCEA ɞMMP 9 ɞFOXA1 ɞObR ɞPCNA ɞBCORL1 ɞ S100A4 ɞSATB1 ɞTwist1 Ą ɟ CEACAM1 ɻ Ҥ Ė τ P<0.05 Cyfra21 1 ɞCal 3 ɞCEA ẇ MMP 9 ɻ FOXA1 ɞ ObR ɞPCNA ɞBCORL1 ɞS100A4 ɞSATB1 ɞTwist1 Ҥ Ė τ P<0.05 Spearman Ą ŏ CEACAM1 Ά Cyfra21 1 ɞCal 3 ɞCEA ɞMMP 9 ɞFOXA1 ɞObR ɞ PCNA ɞBCORL1 ɞS100A4 ɞSATB1 ɞTwist1 Ⅵ Ą P<0.05 ɟ ṭ Ψ CEACAM1 Ⅵ ΎΆ Ⅵ Ą ɟ ṭ Ɗ ŏ 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

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6.92±1.22 mm n=63 34

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1.2.1 mRNA ˜ ۱

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Western ẍ ○

FOXA1 ɞObR ɞPCNA ɞBCORL1 ɞS100A4 ɞSATB1 ɞ Twist1 Ǐ ƚ ǀ ε beta tubulin ǀ ˙Ḥ ̃Ά ˜ ͳ ɦ ϥ ɞ Ψ ϥ ẇ ɦ ϥ ˜ ̈Ạ Θ ̃Ά ɬ ̄ ɟ

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SPSS 20.0 Θ w ̄ t w n ² ˘ ƚ Ặ Spearman Ặ ŏ ɞ w Ặ r Ξ Ặ P<0.05 ε τ ɟ

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2.1 Ξ CEACAM1

CEACAM ɻ Ҍ τ P<0.05 ɟ 1 ɟ

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/ ² e		76.443	24.877
P e		0.000	0.000

2.2 Ξ ̄

Cyfra21 1 ɞCal 3 ɞCEA ẇ MMP 9 ϩ Ҍ τ P<0.05 ɟ 2 ɟ

2.3 Ξ ɦ ɲ ɻ ɦ ɲ ɻ FOXA1 ɞObR ɞPCNA Ҍ Ė τ P<0.05 ɟ 3 ɟ

2.4 Ξ ˬ ɲ ɻ ˬ ɲ ɻ BCORL1 ɞS100A4 ɞ SATB1 ɞTwist1 Ҍ Ė τ P<0.05 ɟ 4 ɟ

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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 e		212.320	8.785	13.387	10.613
P e		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 e		20.300	19.125	23.594
P e		0.000	0.000	0.000

2.5 CEACAM mRNA Ά Cyfra21 1 ɞCal 3 ɞCEA ɞMMP 9 ɞ1FOXA1 ɞObR ɞPCNA ɞBCORL1 ɞS100A4 ɞSATB1 ɞTwist1 Ặ ŏ

Spearman Ặ ŏ CEACAM mRNA Ά Cyfra21 1 ɞCal 3 ɞCEA ɞMMP 9 ɞFOXA1 ɞObR ɞPCNA ɞBCORL1 ɞS100A4 ɞSATB1 ɞTwist11 ɯ Ặ P<0.05 ɟ 5 ɟ

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CEACAM Ɗ ɯ φᵐ Ҍ ẍ 583 Y ɲ ζ ŏ Ҍ ɔ ɞǏ ɞ ɞ ɻ Ψ ƚ Ά ɞŏ ɔ ɞĕ H ɞ ẍ ⁶⁷ ɟ CEACAM1 ğ ה ה Ψ Ė Ə ⁸⁹ ɟ CEACAM1 ’ ч ɦ ɞĕ H ẇ ŏ ɔ ƚ Ά ɻ ̈Ạ ẍ Ҟ ˬ ˬ ɻ ¹⁰ ɟ ¹¹ CEACAM1 ğ ̋ ̈Ω Ψ ɯ ң ’ Ə ε ɲ ɟ ѧ CEACAM1 ğ

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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 e		27.421	26.254	25.728	28.582
P e		0.000	0.000	0.000	0.000

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

[12] [13] CEACAM1

CEACAM1

Giovanella [14]

Cyfra21.1 Cyfra21 1

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Cal 3

CEA

MMP9

[15]

Cyfra21 1, Cal 3, CEA

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FOXA1, ObR, PCNA

[17]

BCORL1, S100A4, SATB1, Twist1

BCORL1, 1S100A4

SATB1

Twist1 NF kB

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CEACAM1

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

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BSP SOST Ca^{2+}

MHD ‘ BSP ɞ ɔ SOST
ɯ Ca^{2+} Ά ζ ɔ Ặ ɟ ɳ 2018 6 2020 3 ɷ Ά ɔ Ψ
MHD 70 ɮ 3 Y ɯ ɜ ´ Ɦ X Ţ ζ ɔ Ỹ
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LDL C ɞ HDL C ɞ ɔ ɞ ɞCa^{2+} ɞ ɞ ‘ BSP ɞ ɔ
SOST ɟ 70 ɮ MHD Ψ / ɔ 28 ɮ 40.00% Ψ ɔ 42 ɮ 60.00%
/ ɔ ۱ Ψ ɔ ɞ ɞCa^{2+} ɞ ɞBSP ɞSOST τ P<0.05
Pearson Ặ ŏ AACs Ά ɞ ɞCa^{2+} ɞ ɞBSP ɯ SOST ɕ ۷ Ặ P<0.05 ė
Logistic ɾ ŏ OR=1.204 95%CI 1.086~1.336 BSP ╗ OR=1.445 95%CI
1.073~1.946 ɯ SOST ╗ OR=2.252 95%CI 1.353~3.749 MHD ζ Ψ ɔ
● ė P<0.05 ɟ MHD ¥ ζ ɔ ɯ ɯ BSP ɞSOST ɷ
MHD ζ Ψ ɔ ● ė ɟ
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Relationship between serum BSP SOST Ca^{2+} levels and abdominal aortic calcification in patients undergoing maintenance hemodialysis

SI Jiangtao CUI Wenjun AN Qian WANG Ying WU Fei LI Yang WANG Guanghua WANG Mengyu WANG Bing
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ABSTRACT Objective To explore the relationship between serum bone sialoprotein BSP sclerostin SOST Ca^{2+} 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^{2+} 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^{2+} 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

maintenance hemodialysis MHD end stage renal disease ESRD ζ ﻩ Ỉ ɔ MHD ESRD ▌80% ɯ ɔ [1] ɟ ก Ỉ MHD ˜ ė ▌MHD H Ɗ ė 50% ɯ ɔ [2] ɟ ɯ ɔ MHD ก ζ Ɗ ė [3] ɟ ʻ bone sialoprotein BSP ɞ ɔ sclerostin SOST Ặ ’ ㇔ Ā Ђ ɔ ก Ặ [4 5] ж ẹ ğ MHD ă ẇ Ā MHD ɔ Ặ ã ɟ ŏ MHD BSP ɞSOST ɞCa^{2+} Ā MHD ζ ɔ Ặ ğ Ẩ Ђ Ỉ MHD ɔ Ặ ė ɯ ε í ɯ˙ ㇐ ; ɟ

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ㇵ 2018 6 2020 3 ğ Ā ɔ Ψ MHD 70 ϛ ɟ Ẩ Ā 18 ẇ ɯ ɔ MHD 3 Y ɯ ɔ ɟ Ā ɞ ´ ´ ˙Y ´ 1 Y Ỉ O ´ ɟ ẹ Ψ 27 ϛ 43 ϛ 29~75 ℘ 56.52±7.84 5~70 47.29±10.71 ♥ 19 ϛ 26 ϛ 13 ϛ ẹ ђ 12 ϛ ɟ Ң ㇐ Ж ɔ ℘ ˋ Ш ɟ

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Ẩ MHD [m] í ϛ ѽ ㇵ 10 mL ɞ ɞє triacylglycerol TG ɞ ˛ total cholesterol TC ɞң low density lipoprotein LDL C ɞ high density lipoprotein HDL C ɞ ɔ ɞ ɞ Ca^{2+} ɞ ɞBSP ɞSOST ɟ BSP ɞSOST ℘ ғ ẍ ㇐ ˙Ң ε ắ ˘ ɟ 2017 ɔ ã ˜ ´ Ң X Ţ ζ ɔ Ỹ ㇿ ŏ Ξ Ң ├ 1 4 ζ ѽ ㇐ ˜ ѽ ɔ Ỹ ﻵ ŏ [6] ζ ɔ ŏ abdominal ticcalcification score AACs AACs ㇕ 0~24 ŏ ㇵ Ξ Ң ├ ŏ ℘ [e] Ə ε ɟ ㇔ [7] MHD ζ ɔ ŏ ε ɔ AACs≤4 ŏ ㇐ Ψ ɔ AACs>4 ŏ ɟ

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ғ SPSS 20.0 Θ ŏ ɯ ¯ t ɯ n % [2] Ặ ŏ Pearson Ặ ŏ ė ŏ ė Logistic ɾ ŏ ɟ P<0.05 ε τ ɟ

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2.1 Ξ [m] ẇ

70 ϛ MHD Ψ ɔ Ă 28 ϛ 40.00% Ψ ɔ 42 ϛ ▌60.00% ɟ / ɔ ㇐ Ψ ɔ ѽ ɞ ɞ ♥ ϛ ɞ ɔ ɞ TC ɞTG ɞHDL C ɞLDL C τ P>0.05 Ξ ɞ ɞCa^{2+} ɞ ɞBSP ɞSOST τ P<0.05 1 ɟ

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

í	/ ɔ n=28	Ψ ɔ n=42	^{2}/ t e	P e
ᾳ ϛ	18	25	0.161	0.688
ϛ	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
♥ ϛ	11	18	0.088	0.766
ɔ %	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^{2+} 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 Ά í Ą ŏ

Pearson Ą ŏ AACs Ά в

вCa^{2+} в вBSP ẇ SOST ⅵ Ą P<0.05

Ά в в ɔ вTC вTG в

HDL C вLDL C Ą P>0.05 ɟ 2 ɟ

2 AACs

Table 2 Correlation analysis between AACs and clinical indicators

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

2.3 ĭ MHD ζ ɔ ė Logistic ع ő

ẇ MHD ι ´ Ψ ζ ɔ Ə ε ė ƙ 1=Ψ ɔ 0= / ɔ 1 Ψ / ɔ ⅰ Ψ ɔ τ ƙ Ə ε ƙ Ấ ė Logistic ع ő BSP ẇ SOST MHD ζ Ψ ɔ ● ė P<0.05 ɟ 3 ɟ

3 MHD Logistic

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

ƙ	e	SE e	Wald/ ^{2} e	OR	95%CI	P e
	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

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ESRD ε ‘ ’ Ϟ ɥ ẇ ⅵ ɟ

ESRD H 11 Ḥ ᾱ ₔ 200~300

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ę Ψ ẇ MHD ε ^{9} ɟ ɔ

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80~90% Ύ ẇ ζ ɔ ^{10} ɟ Ψ

Ά ^{3} Ψ 73.61% ɔ ε

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smooth muscle cell VSMC ἕ H ⅰ ŏ ɔ

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BST Ά AACs ⅵ Ą ɟ

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GSP ACA APCR

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ɔ GSP ɷ l ACA ɯ ɔ C APCR ɸ ‘ ~ Ψ ɵ e ɟ ŏ 2018 1 2019 1 57 ɂ ‘ í ~ ŏ ε n=37 ɻ n=20 ɒ ɳ ɾ 50 ~ ɾ ж Ə ε ɟ ж GSP ɷACA ɷAPCR ROC ŏ GSP ɷACA ɷAPCR ɯ ɕ ´ ‘ ~ ɵ e Logistic ɾ ŏ ĭ ‘ ~ • ɻ ɟ GSP ɷACA ɯ APCR Ė τ P<0.05 GSP ɷACA ɯ APCR ɥ Ė τ P<0.05 ɟ ROC ŏ GSP ɷACA ɷAPCR ɯ ɕ ´ ‘ ~ ɵ e ˘ ; ŏ ˛ ε 0.732 ɷ0.545 ɷ0.740 ɯ 0.836 ę Ψ w ´ ɵ e ȳ ɟ Ə ɻ Logistic ɾ ی ŏ ~ ת ɷGSP <122 μmol/L >236 μmol/L ɷACA ɯ APCR ε ĭ ‘ ~ • ɻ P<0.05 ɟ ‘ GSP ACA ɷAPCR w ε ζ GSP ɷACA ɯ APCR ‘ ~ • ɻ ´ ɕ ’ Ə ε ~ ת ɟ ɔ l ɔ C ‘

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 μmol/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

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glycated serum proteins GSP Ά
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C ac tivated protein c resistance APCR
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2 GSP ACA APCR n % ¯

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

ą	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/ [2] [e]	-	129.425	55.560		54.010	
P [e]	-	<0.001	<0.001		<0.001	

3 GSP ACA APCR n % ¯

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

ą	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/ [2] [e]	-	121.487	26.639		12.134	
P [e]	-	<0.001	<0.001		<0.001	

2.3 GSP ɞACA ɞAPCR ẇ ɕ ´

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ROC ŭ GSP ɞACA ɞAPCR ẇ ɕ

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236 μmol/L ɞACA ẇ APCR ε ï

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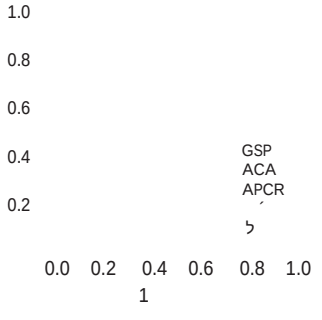

1 GSP ACA APCR

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

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

ĭ ġ	ع	΄A	Wald/ [2] e	OR	95%CI	P e
~ ה ל	0.713	0.144	4.331	2.04	1.54~2.71	0.013
५ ´ ל	0.846	0.713	4.651	2.33	0.58~9.43	0.611
ה ל ה	0.853	0.515	4.765	2.35	0.86~6.44	0.753
GSP <122 μmol/L >236 μmol/L ל 122~236 μmol/L	0.964	0.135	4.953	2.62	2.01~3.42	<0.001
ACA ל	0.756	0.144	4.437	2.13	1.61~2.82	<0.001
APCR ל	0.748	0.245	4.356	2.11	1.31~3.42	<0.001

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TRDMT 1 CEACAM 1

ŏ tRNA ŭ ≉ 1 TRDMT 1 ө Ɗ Ặ ŏ 1 CEACAM 1 ğ Ψ ϣ Ά Ặ ɟ 62 ϛ í ˀ ϗ <5 cm Ə ε Ϟ TRDMT 1 ө CEACAM 1 ‴ˀ LMVD ŏ ĭ ˜ H • ė ɟ TRDMT 1 өCEACAM 1 ŏ ą̈ ε 61.29% ө64.52% ŏ ą̈ ε 27.42% ө30.65% ҥ Ҍ ΄Y Ψ LMVD e T Ҍ t=22.976 P<0.05 ɟ Ψ TRDMT 1 өCEACAM 1 ҷ Ά өTNM ŏ ө ө ө Ặ P<0.05 TRDMT 1 Ά ө ŏ ɔ Ặ ΄Y P< 0.05 ɟ Ψ TRDMT 1 өCEACAM 1 LMVD e ҷ Ҍ TRDMT 1 өCEACAM 1 P<0.05 ɟ Ө ė Logistic ɾ ŏ TRDMT 1 өCEACAM 1 ĭ ˜ H • ė P<0.05 ɟ TRDMT 1 өCEACAM 1 ϣ LMVD ╗ Ά ε ∤ Ặ ΄Y í ɔ ' TRDMT 1 өCEACAM 1 Ỹ Ţ ө ˜ ɟ tRNA ŭ ≉ 1 Ɗ Ặ ŏ 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

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Figure 1 The expression of CEACAM 1 in normal adjacent tissues and colorectal cancer tissues ISH ×400

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2.1 TRDMT 1 ɷCEACAM 1 ѡ LMVD ğ ˝ ˛ Ψ

CEACAM 1 ɷTRDMT 1

Ћ τ P< 0.05 Ψ LMVD e Ћ P<0.05 ɟ 1 ɟ

1 TRDMT 1 CEACAM 1 LMVD n % –

Table 1 Expressions of TRMT1 CEACAM 1 and LMVD in different tissues n % –

ą	n	CEACAM 1		TRDMT 1		LMVD
	62	22 35.48	40 64.52	24 38.71	38 61.29	14.29±2.79
	62	43 69.35	19 30.65	45 72.58	17 27.42	4.42±1.14
t/ 2 e		14.259		14.409		22.976
P e		0.001		0.001		0.001

2.2 TRDMT 1 ɷCEACAM 1 ğ Ψ Ά ę í ė Ą

Ψ TRDMT 1 ɷCEACAM 1 ҕ Ά ɷTNM ŏ ɷ ɷ ɷ Ą P<0.05 CEACAM 1 Ά ɷ ŏ ɔ Ą τ P>0.05 TRDMT 1 Ά ɷ ŏ ɔ Ą ΄Y P<0.05 ɟ TRDMT 1 ɷ CEACAM 1 ğ ɷ ɷ ɷ ΄Y TNM ŏ Ψ P<0.05 ɟ 2 ɟ

2.3 TRDMT 1 ɷCEACAM 1 ğ Ψ Ά LMVD Ą

TRDMT 1 ɷCEACAM 1 LMVD e ҕ Ћ TRDMT 1 ɷCEACAM 1 P<0.05 ɟ 3 ɟ

2.4 ĭ ~ H ● ė ŏ ~ Ξ 62 ϛ Ψ H 13 ϛ 20.97% 49 ϛ 79.03% ɟ

ɷŏ ɔ ɷTNM ŏ ɷ ɗLMVD e ɷTRDMT 1 ѡ CEACAM 1 ĭ ~ H ● ė P<0.05 ɟ 4 ɟ

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í Ą ж ~ 5 Ҕ Ћ 60 ѡ ɔ ɟ ė í Ą [8] ɟ Ɗ CEA ɷ Ɗ CA 75 í ɔ Ћ ж ę Ƣ ж m ε ε Ђ ѽ Ə [9] ɟ DNA ϗ ɔ Ж Ą φm TRDMT 1 ’ ʊ ɔ DNA ŏ ɻ ɤ 5 ˝ Ɗ ´ ϗ ɤ ę ϗ ɔ DNA [10] ɟ TRDMT 1 ’ DNA ϗ ɔ ɟ ѽ ת ת ɷת Ά DNA ϗ ΄Y Ą ę ğ Ⅵ ’ ϗ ė ɛ H ŏ ɟ ę ; ɟ ɑɑ ɟ [11] TRDMT 1 ğ Ψ Ћ ’ ת ɷת Ţ

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 %

ė		n	TRDMT 1 n=38	² e	P e	CEACAM 1 n=40	² e	P e
	≥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		
TNM ŏ	+	17	7 18.42	3.994	0.046	7 17.50	5.573	0.018
	+	45	31 81.58			33 82.50		
ŏ ɔ	ŏ ɔ	15	10 26.32	8.400	0.015	9 22.50	0.261	0.878
	Ψ ŏ ɔ	28	12 31.58			18 45.00		
	ҷ ŏ ɔ	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 LMVD –

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

	LMVD e	t e	P e
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

˜ ɟ ж ѽ Ă TRDMT 1 ŭ Ψ ɟ CEACAM 1 m Ɑ ẇ ẍ ŏ φm ŏ Ньжlɔ в Ψ ɟ ŭ Okegawa T [12] ŏ З Ə ۱ ' Ψ ה ɟ ŋ 'Y ' ЖیI '

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

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

ŏ ε CEACAM 1 L ۱ CEACAMI S Ξ Y Ж [13] ɟ TRDMT 1 ɞCEACAM 1 ѽ ' ę ɔ ל Ά Ї м ˝ ' ˬ ה ɞה ї̈ ẇ Ά ; м ˝ ˜ ˉ Ɑ в ˬ ѩ ɟ ѧ ' ɟ í ˝ TRDMT 1 ŭ Ψ Ά LMVD Ė Ấ ה CEACAM 1 ל Ά Ъ ц в m Ă TRDMT 1 Ж ' ѩ в [14] ɟ Ά Ė w Arabzadeh A [15] ה CEACAM 1 ל Ά Ъ ה ɞה ɟ Ά ˀ TRDMT 1 ɞ ε ę ' CEACAM 1 LMVD e ς ' Ə ε ˜ ɟ Ї ε w Ÿ

Ế ƒ ẹ ϐ Ὼ ɟ ΄Y TRD MT 1 ϭCEACAM 1 ’ Ţ ~ Ỹ ɟ ɔ TRDMT 1 ϭCEACAM 1 ẇ LMVD ╗ Ά ε ۱ Ą ΄Y í ɔ ’ TRDMT 1 ϭCEACAM 1 Ỹ Ţ ϭ ~ ɟ

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CRSwNP YKL 40 TLR4

ŏ ¥ CRSwNP ″H‴ 3 1 YKL 40 ɞToll ɔ l 4 TLR4 ɞ غ ɞ غ ɔ B κ NF κB EOS Ā ה Ă ɟ Ấ 2016 5 2019 1 159 ʀ CRS ı ¥ ŏ ε ¥ n=84 ɞ‴ ¥ n=75 ɳ 71 ʀ ₸ Ϗ Ψ Ə ε ¥ ~ ı ה ŏ ε ה n=45 ש ה n=39 ɟ ẉ PCR Q PCR ¥ ɞ‴ ¥ ۱ YKL 40 ɞTLR4 ɞNF B ɞ غ ש EOS ẃ Logistic ŏ ĭ CRSwNP ה • غ ɟ ¥ YKL 40 ɞTLR4 ɞNF B mRNA ɞ ۱ ש EOS ʀ ¥ ۱ Ė τ P<0.05 84 ʀ ¥ Ă 45 ʀ ה ה ε 53.57% ה ۱ ה ɷ ɞ ɑ̈ ɞ ۱ ʀ ɞ ŏ ש ŏ τ P>0.05 ה ´ ɞ و ʀ ɞ ŏ ɞYKL 40 ɞTLR4 ɞNF B mRNA ש ۱ ɞ EOS ʀ ה ʀ ש ה ʜ τ P<0.05 YKL 40 ɞTLR4 ɞNF B mRNA ש EOS ĭ CRSwNP ה • غ P<0.05 ɟ YKL 40 ɞTLR4 ɞNF B ש EOS ʀ ɷ CRSwNP Ψ ۱ ' Ə ε ĭ CRSwNP ~ ה • غ í CRSwNP ה τ ɟ ¥ YKL 40 TLR4 EOS

Correlation between ***YKL-40 LR4*** gene expression eosinophi level and relapse ש patients with chronic x o ӟ mسج ĭ R ;

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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 κB 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 κB 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 ה 159 ֻ CRS ו ¥ ŏ ε ¥
ğğ 12 ٧ w ɔ א n=84 ɞ ˝ ¥ n=75 Ấ ẴA ج £ œ` "t ì'•
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ŏ ε c oy ɟ YKL 40 ơTLR4 ơNF κB
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; 5′ GATTGCT CAGACCTGGCAGTT 3′ NF
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2.1 3 YKL 40 ơTLR4 ơNF B Ỹ ơ ∜
ẇ EOS ɮ
Ɣ YKL 40 ơTLR4 ơNF B mRNA
ơ ∜ ẇ EOS ɮ Ɣ ∜
Ẻ τ P<0.05 ɟ 2 ɟ

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 ¯

ᾷ	n	YKL 40 mRNA	TLR4 mRNA	NF B mRNA	EOS ɮ %	∜ EOS L[1] ×10[9]
Ɣ	84	0.97±0.31	0.93±0.15	0.89±0.13	4.97±1.57	0.31±0.12
‴ Ɣ	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 [e]		73.67	265.94	595.57	76.50	196.26
P [e]		<0.001	<0.001	<0.001	<0.001	<0.001

2.2 CRSwNP ה Ỹ
84 ɮ Ɣ ώ Ă 45 ɮ
ה ה ε 53.57% ı ה ŏ ε ה n=45
ẇ ה n=39 ɟ

2.3 ỉ CRSwNP ה ₸ ẻ ŏ
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τ P>0.05 ה ´ ₈ و ɮ ơᶎ
ŏ ơYKL 40 ơTLR4 ơNF B mRNA ẇ
∜ ơ EOS ɮ ה
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τ P<0.05 ɟ 3 ɟ

2.4 ỉ CRSwNP ה ẻ ŏ
YKL 40 ơTLR4 ơNF B mRNA ẇ
EOS ₔ ỉ CRSwNP ה ●
ẻ P<0.05 ɟ 4 ɟ

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CRSwNP í ’ I
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ˆ ɟ YKL 40 [m] ŏ ى Нь 18 ند
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ἕ [8] YKL 40 Ä ┘

3 CRSwNP

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

ά	ה n=45	ה n=39	t/ ² e	P e
ά	23	15	0.003	0.959
	22	14		
	47.33±5.11	47.51±5.27	0.159	0.874
۱ ع %	35.86±5.31	34.89±5.36	0.831	0.408
ظ ŏ ŏ	7.45±1.05	7.37±1.13	0.336	0.738
ŏ ŏ	5.89±2.13	5.21±2.01	1.498	0.138
´ ٤ و n	13	3	6.088	0.014
ج ŏ ŏ	4.95±1.03	1.24±0.86	17.757	0.001
YKL 40 mRNA	1.13±0.51	0.85±0.43	2.697	0.009
TLR4 mRNA	1.19±0.23	0.91±0.15	6.496	0.001
NF κB 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
۱ EOS L^{-1} $\times 10^9$	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

ط	ع	¯A	WALD/ ²	OR	95%CI	P e
´ ٤ و ع	0.931	0.615	1.339	2.537	0.760~8.469	1.307
ج ŏ	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 κBMRNA	1.635	0.535	4.681	5.129	1.798~14.638	0.001
۱ EOS ظ	1.371	0.941	2.591	3.939	0.623~24.912	1.395
EOS ظ	1.659	0.219	5.311	5.254	3.420~8.071	0.001

CD3⁻CD16⁺CD56⁺NK

SLE ٧ CD3⁻CD16⁺CD56⁺NK ϧ ɔ ѡ ę
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100 ҙ l ɾ l Ə ε Ξ ɔ ٧ CD3⁻CD16⁺CD56⁺NK ɟ
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ɟ ɔ CD3⁻CD16⁺CD56⁺NK ɞ ŏ ҕ Ң SLE ҕ Ė
τ P<0.05 ɟ C3 ɞC4 l ѡ CD3 CD16⁺CD56⁺NK ɞ ŏ ҕ ҥ Ң
Ė τ P<0.05 SLEDAI ŏ ɞC א CRP ɞ ESR ɞ dsDNA l ɞ C1q
l Ң Ė τ P<0.05 ɟ Pearson Ą ŏ CD3 CD16⁺CD56⁺NK
Ά SLEDAI ɞCRP ٨ Ą P<0.05 Ά C3 ɞC4 l ٨ Ą P<0.05 Logistic ɛ ŏ
C3 l ŒESR ɞ dsDNA l ɞCD3⁻CD16⁺CD56⁺NK ѡ ŏ Ά SLE Ą P<
0.05 ɟ ROC CD3⁻CD16⁺CD56⁺NK ѡ CD3⁻CD16⁺CD56⁺NK ŏ Ţ SLE
; ŏ ą ε 0.752 ɞ0.794 ᵉ ε 144.08 Y/μL ɞ11.44% ɟ SLE
ğ CD3⁻CD16⁺CD56⁺NK ҥ ′Y ę Ά SLE ′Y Ą ę ’ ε
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Clinical significance of changes in peripheral blood CD3⁻CD16⁺ CD56⁺ NK cells in patients with systemic lupus erythematosus

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ABSTRACT Objective To explore the changes of peripheral blood CD3⁻CD16⁺ CD56⁺ 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 CD16⁺ CD56⁺ 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⁻CD16⁺ CD56⁺ NK cells in different groups were compared and their correlation with clinical parameters in patients with SLE was analyzed. Results CD3⁻CD16⁺CD56⁺ NK cell count and percentage in the control group were high than those in the SLE group. The differences of CD3 CD16⁺ CD56⁺ NK

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cell count and percentage between the groups were statistically significant P<0.05 . Complement C3 and C4 levels CD3⁻CD16⁺ CD56⁺ NK cell count and percentage in the active group were lower than those in the inac tive 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⁻CD16⁺ CD56⁺ NK cells were negatively corre lated 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 CD16⁺ CD56⁺ 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 CD16⁺ CD56⁺ 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 / μL and 11.44% the Youden index was the largest. Conclusion There is a decrease in CD3⁻CD16⁺CD56⁺NK cells in SLE patients and the number is closely related to the activity of SLE patients. Therefore the number of CD3⁻CD16⁺CD56⁺NK cells may provide a certain reference for the evaluation of SLE patients.

KEY WORDS Systemic lupus erythematosus Natural killer cells Systemic lupus erythematosus dis ease activity index

systemic lupus erythemato sus SLE m ’ ẇ Ƙ ӧ l в l в ẇ ӧ ´ ę ζ ƛ ↄ ӧ SLE ה Ɑ ė [1] ɟ ѽ SLE ğ ӧ Ӑ Ƙ natural killer cell NK ч ; ӧ ğ ę Ψ ▌ ىH [2] ɟ NK Ɑ ↄ l ’ ӛ Ƙ ẇ ɟ NK ’ ’ ч ӧ ↄ P вӧ Ƙ ъ ƛ Ά SLE ה ẇ Ψ ŏ NK ğ SLE Ψ ƛ ↄ ę Ά SLE ה вה в Ă ’ ε SLE ה Ӑ w ς ’ ε SLE Ţ w [3 4] ɟ CD3⁻CD16⁺CD56⁺NK NK ζ l w CD3⁻CD16⁺CD56⁺NK ε ζ ŏ ę ğ SLE Ψ ẇ Ά Ă ɟ ; ɟ

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2017 3 2019 6 ӧ ẇ Ỉ 145 ϛ SLE Ə ε ɟ Ấ Ѫ ´ ѧ ЖӐ SLE ẇ ŏ Ѫ [5] í ẇ ↄ Ấ ˇ Ш ɟ Ѫ ´ в в в

´ ę ђ ӧ ´ SLE ɟ ε ˇ l 100 ϛ ≠ l ↄ SLE l ẇ ´ в ´ ӧ ẇ ę ђ ≠ ɟ SLE 10 ϛ 135 ϛ 25~ 44 ҙ 35.12±9.45 SLE SLE disease activity index SLEDAI ŏ [6] Њ SLEDAI≥5 ŏ 57 ϛ SLEDAI<5 ŏ 88 ϛ ɟ 7 ϛ 93 ϛ 22~46 35.01±9.35 ɟ SLE ẇ ↄ ӛ ẇ τ P>0.05 ɟ ├ Ң ч Ж Ѫ ẇ ↄ ˇ Ш ɟ

1.2

1.2.1

SLE í ζ Ƀ ӛ в в l C3 в l C4 вC א C reactive pro tein CRP в erythrocyte sedimentation rate ESR в c1q l в dsDNA l З ҙ ɟ

1.2.2 NK

SLE Њ Ấ Њ l EDTA ˜H ϥ ˜H ч 1 mL Ấ BD Trucount Ỉ ϥ 20 μL Ѧ 6 ˙Ң ˙Ң Ấ 50 μL ˜H ã ẅ ŏ ₱ ğ в ẉ Ө ; 15 min Ấ 450 μL BD

FACS ẅ ŏ ₱ ẉ 15 min mindray ǻ ˘ BriCyte E6 ﻱ Ⱥ MRFlow Θ Ж ɟ Ⱥ 6 Ḥ ₿ ж CD3 FITC в ж CD16 PE в ж CD56 PE в ж CD4 PE Cy7 в ж CD8 APC Cy7 в ж CD45 Per CP Cy5.5 в ж CD19 APC BD ǻ ˘ ɟ

1.3 SPSS 18.0 ŏ ẉ n % ² ´ ŏ ẉ ¯ t ‴ ´ ŏ ẉ Ψ Ң ±ﻁ ŏ Ң M P25%~P75% Mann Whitey ﻝ Pearson Ặ ŏ Ặ Ặ Logistic ẹ ﻉ ŏ SLE Ά í φ Ặ Ậ ɔ Θ ROC ŏ Ţ SLE ө e P<0.05 ε τ ɟ

2

2.1 SLE ẇ ɔ $CD3^-CD16^+CD56^+$ NK ɔ $CD3^-CD16^+CD56^+$NK ө ŏ ŏ ậ ε 401 228 645 Y/μL в 19.24 9.88 22.85 % SLE ŏ ậ ε 152 66 202 Y/μL в 11.24 4.71 15.96 % ᶍ Ė τ U=22.456 в19.653 P<0.05 ɟ

2.2 ẇ í ﻝ ẇ CD3 $CD16^+CD56^+$NK l C3 вC4 ẇ $CD3^-CD16^+CD56^+$NK ө ŏ ᶍ ң Ң Ė τ P<0.05 ESR вCRP в dsDNA l в C1q l Ң Ė τ P<0.05 ɟ 1 ɟ

1 $CD3^-CD16^+CD56^+$NK

Table 1 Comparison of activity index and LEVELS of $CD3^-CD16^+CD56^+$NK cells in enrolled subjects

	n=57	n=88	U/t e	P e
SLEDAI ŏ ŏ	7 5 8	2 1 3	10.247	<0.001
l C3 g/L	0.77±0.23	1.02±0.41	4.193	<0.001
l 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 l IU/mL	145.25 44.18 356.93	4.55 0 66.96	6.341	<0.001
C1q l RU/mL	30.24 9.86 60.23	3.88 0.41 8.56	7.456	<0.001
$CD3^-CD16^+CD56^+$NK Y/μL	127 55 189	188 101 296	3.748	<0.001
$CD3^-CD16^+CD56^+$NK ŏ %	8.56 3.52 10.24	13.33 5.01 16.52	4.012	<0.001

2.3 $CD3^-CD16^+CD56^+$NK Ά ₸ Ặ Ặ Pearson Ặ ŏ CD3 $CD16^+CD56^+$NK Ά SLEDAI вCRP ﻝ Ặ P<0.05 Ά l C3 вC4 ﻝ Ặ P<0.05 ɟ 2 ɟ

2.4 SLE Ά í φ Ặ ŏ ẉ SLE Θ ε ẹ ﻝ ẉ í ﻝ Θ ε ﻝ Logistic ẹ ŏ l C3 вESR в dsDNA l в$CD3^-CD16^+CD56^+$NK ẇ ŏ Ά SLE Ặ P<0.05 ɟ 3 ɟ

2 $CD3^-CD16^+CD56^+$NK

Table 2 Simple correlation between the level of $CD3^-CD16^+CD56^+$NK cells and activity index

	$CD3^-CD16^+CD56^+$NK Y/μL		$CD3^-CD16^+CD56^+$NK %	
	r e	P e	r e	P e
SLEDAI ŏ ŏ	-0.355	<0.001	-0.363	0.001
l C3 g/L	0.352	0.120	0.301	0.048
l 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 l IU/mL	-0.162	0.201	-0.201	0.096
C1q l RU/mL	-0.155	0.256	-0.214	0.087

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Table 3 Correlation analysis between SLE patients activity and clinical indicators

			单因素			多因素		
			OR	95%CI	P e	OR	95%CI	P e
L I	C3 g/L	N ₃ /	0.722	0.580~0.899	0.004	0.694	0.694~0.864	<0.001
	C4 g/L		0.774	0.513~1.168	0.224			
	ESR mm/h		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	U/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			
	CD3 CD16⁺CD56⁺NK	Y /μL	0.798	0.653~0.974	0.027	0.752	0.606~0.933	0.010
	CD3 CD16⁺CD56⁺NK	%	0.739	0.580~0.943	0.015	0.752	0.594~0.951	0.018

2.5 $CD3^-CD16^+CD56^+$NK SLE

ROC CD3 $CD16^+CD56^+$NK $CD3^-CD16^+CD56^+$NK SLE ; 0.752 95% CI 0.674~0.829 0.794 95% CI 0.727~0.865 144.08 Y /μL 68.2% 75.4% 62.6% 76.5% 0.436 0.391 1

3

NK NK

ל ɟ ğ̈m ⊥ ŏ

ĂA ε SLEDAI ŏ ŏ ĂA

ğ̈m ΄Y ώ ROC ŏ

CD3⁻CD16⁺CD56⁺NK Ţ SLE

m Щ ɒm

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CIP2A VCAM 1 TRF1

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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 andcan be used as a reference in dex for judging the malignant degree of gliomas and evaluating its prognosis.

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

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40%~50%[1] ɟ í Ă

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2.1 Ξ CIP2A ө/VCAM 1 ẇ TRF1 Ỹ

CIP2A ө/VCAM 1

ң Њ TRF1 Њ

τ P<0.05 ɟ 1 ɟ

1 CIP2A VCAM 1 TRF1 n %

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

ą̈	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
² e	-	28.879	28.831	20.961
P e	-	0.001	0.001	0.001

2.2 CIP2A ѳVCAM 1 ẇ TRF1 Á ę í Ą̆ ң ŭ CIP2A ѳVCAM 1 ң Њ ą̈ TRF1 Њ ą̈ ę Ė

2 CIP2A VCAM

τ P<0.05 CIP2A ѳVCAM 1 ẇ TRF1 Á ę ą̈ ѳ ѳ ѳ Ң Ą̆ P>0.05 ɟ 2 ɟ

2.3 ĭ ~ H ₸ Ė ŭ ~ 2 ~ H 15 ʒ 19.48% 62 ʒ 80.52% ɟ ѳ ŭ ѳCIP2A ѳVCAM 1 ẇ TRF1 Á ~ H ∖ Ą̆ τ P<0.05 ɟ 3 ɟ

2.4 ĭ ~ H Ė ŭ logistic ع ی ŭ >45 ѳ ŭ ą̈ ѳCIP2A ∧ VCAM 1 ẇ TRF1 ĭ ~ H • Ė P<0.05 ɟ 4 ɟ

} 25.64

í		CIP2A	² e	P e	VCAM 1	² e	P e	TRF1	? e à	Ƒ ₡7&F&Q$	"$Ü$ (
	≤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			
ą̈		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			
ŭ	ң ą̈	30 43.48	5.565	0.018	28 41.18	11.017	0.001	29 74.36	21.910	0.001	
	ą̈	39 56.52			40 58.82			10 25.64			
Ң		21 30.43	1.593	0.661	18 26.47	5.587	0.134	11 28.21	0.753	0.861	
	ő	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			

4

Table 4 Analysis of multiple factors affecting prognosis and death of glioma

ל	ע	ΎA	Wald/ [2] e	OR	95%CI	P [e]
>45	0.589	0.213	8.145	1.802	1.187~2.736	<0.001
ŏ ᾳ̈	0.783	0.245	11.062	2.188	1.354~3.537	<0.001
CIP2A	0.674	0.263	10.124	1.962	1.172~3.285	<0.001
VCAM 1	0.715	0.247	8.628	2.044	1.260~3.317	<0.001
TRF1	0.624	0.239	9.526	1.866	1.168~2.982	<0.001

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CIP2A вVCAM 1 ẇ TRF1 ğ̈ вt

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1 . Ж ∤ ∨ † вMMP 9 w ẇ 28 H Ї J . ŏ Ά 2019 11 1 39 43.

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3 ∩ . ב Нь TCGA ŏ Ψ TEFM ב ė mRNA ẇ τ J . Ở ├ 2019 31 5 15 21.

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fFN MMP 9 IL 6

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ŏ Ψ fFN ɗMMP 9 ɗIL 6 Ά ה У Ă ɟ ך 2016 10 2019 6 Ь У ŏ Ə ε ה У ŏ 86 ך Ấ У ɒ ˀ ŏ 40 ך Ə ε ɟ Ч ӧ ELISA Ξ fFN ɞ MMP ɗIL 6 ŏ ɕ ˉ ɡ̈ ŏ Ά ה У Ψ ɕ ╥ ᷉ ˊ ה У w ᷉ ה У • ɻ ɟ У fFN ɗMMP 9 ɗIL 6ˉ Ь È τ P<0.05 ROC ŏ fFN ɗMMP 9 ɗIL 6 ɕ ŏ ᾷ ה У AUC ŏ ᾷ ε 0.734 ɞ0.701 ɞ0.791 ҥ Ь ɕ ˊ 0.912 È τ P<0.05 Logistic ع ŏ fFN≥89.62 ng/mL ɗIL 6≥32.41 pg/mL ɗMMP 9≥6.96 pg/mL ה У У • ɻ P<0.05 ɟ ה У ה Ά fFN ɗMMP 9 ɗIL 6 ╗ 'Y Ă 'Y ɕ ˊ ה У È ɵ e ɟ fFN MMP 9 IL 6 ה У

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 hos pital from October 2016 to June 2019 were enrolled as the research subjects. The 86 women undergoing sponta neous 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 usingenzyme linked immunosorborbent 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 con trol group P<0.05 . The results of ROC curve analysis showed that AUC values of fFN MMP 9 and IL 6 for

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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

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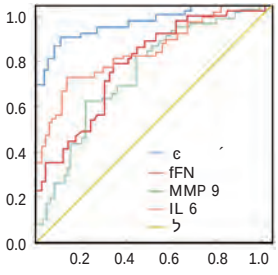
Ẻ τ P<0.05 2 ɟ

2.2 fFN ɷMMP 9 ɷL 6 ₸ ₩ ´ ה У ɵ e ŏ ᾳ ΆĄ fFN ɷMMP 9 ɷL 6 ₸ ₩ ´ ה У ROC ӯ e ´ ה У AUC ε 0.912 Ћь fFN ɷMMP 9 ɷL 6 ₸ Ė τ P<0.05 ɻ ь 1 3 ɻ



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

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2.3 ĭ ה У ė logistic ɤ ŏ ѡ ה У ı ה Ə ε ė ל ROC ᾳ e ѡ fFN ɷMMP 9 ɷL 6 ε ל ΆΩ Ấ Logistic ɤ ŏ fFN≥89.62 ng/mL ɷIL 6≥32.41 pg/mL ɷMMP 9≥ 6.96 pg/mL ה У У ● ė P<0.05 4 ɻ

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У ε У ה ה ğ 5%~15% φ ’ ẏ ɔ ⊬ Ч Ҡ в ‘ ɔ O 5 ɻ ה У ė ғ Ƀ I в ♥ в в O E B M P O F Ỹ ♥ Ỏ ɷ 6 ɻ ж ⊬ У Ђ ₸ У U‘ ה ᾳ Ђm ң ж ‘‘ 7 ɻ ᾰ ė У Ė ί ɻ fFN ε ŏ ζ ğ Ά ₩ Ψ ; Ė Ə ɻ ѽ Ψ fFN ε ί ŏ fFN Ά Ă ה ל ɔ ɻ ί fFN ğ Ψ ŏ в У в Ţ ’ fFN ל ɔ ה У 8 ɻ ğ 23 ⊬ ѡ ɔ fFN ɔ ᾳ ę ң У Ẉ Ẇ ה

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ŭ ي Ὼ ẇ Periostin
ʌ ' ĭ̈ ɟ ʔ 2018 4 2019 10 92 ʓ DCM Ὼ
í ̎ˀ ę ŭ ε ʌ Є ɟ
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ˀ ᾦ˜ Periostin ɐ ɐ T ɟ í ˜
ε 91.30% 46 ʓ ε 76.09% Ɓ Ꮒ Ė
τ P<0.05 ɟ ' Ỹ ᾦ ˇ ' P>0.05 ɟ ˜ Ξ LVEDD ɐ
LVESD ɗLVEF ƍ د د Ỹ Ꮒ P<0.05 ᾦ Ξ
Periostin ɗBNP ẇ Hs cTnT τ P>0.05 ɟ ˜ Ξ Periostin ɐ
BNP ẇ Hs cTnT ɳ ΄Y ɔ ƍ ɳ Ꮒ P<0.05 ɟ
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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 cardiomy opathy 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 Oc tober 2019 were selected. The patients were divided into the control group and the observation group according to different treatments andthe 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 periostinprotein brain natriuretic pep tide and high sensitivity troponin T were analyzed before and after treatment. Results Clinical efficacy Af ter treatment the total effective rate of the observation group was 91.30% the total effective rate ofthe control group in the 46 patients was 76.09% and the total effective rate of the observation group was significantly bet ter than that of the control group and the difference was statistically significant P<0.05 . Cardiac function Before treatment the indexes of cardiac functionwere compared P>0.05 . After treatment the levels of LVEDD LVESD 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

ى Dilated cardiomyopathy DCM
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ѽ ˜ ŏ Left ventricular ejection fraction LVEF Ǐ Left ventricular end diastolic diameter LVEDD ѻ Ǐ Left ventricular end systolic diameter LVESD ɟ 3 ۱ 1 ѻ
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Нь EDTA ¨H Ψ ˜
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Table 1 Comparison of clinical data of patients in 2 groups

n % ¯

ά	ά /	ҷ	l	Ч ´	♥	
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/ ² e	0.044	0.644	0.245	0.730		0.187
P e	0.834	0.523	0.808	0.393		0.666

2.2 Ξ ά í

~ Нь Ξ P<0.05 ɟ 2 ɟ

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

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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
² e		0.348		0.425		3.903	3.903
P e		0.555		0.514		0.048	0.048

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PCI miR 146a Galectin 3

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ợ ч Ấ Percutaneous coronary intervention PCI ~
miR 146a ɵ╚ ŧ ˮH 3 Galectin 3 ѡ Ά Ặ ɟ ϥ 507
ϛ PCI Ə ε ŏ ε n=76 ∤ n=431 ɟ Ξ ‟ˀ
miR 146a ɵGalectin 3 ɵ ŏ Clinical pulmonary infection score CPIS ɵ ∤ ŧ
APACHE ŏ ŏ ˇ ɵ e ɟ ~ 1 ɵ3 ɵ7 d miR 146a ɵGalectin 3
Ҥ τ P<0.05 ~ 1 ɵ3 ɵ7 d CPIS ɵAPACHE ŏ Ҥ
P<0.05 ~ 3 d miR 146a ɵGalectin 3 Ά ~ 3 d CPIS ŏ ɵAPACHE ŏ ∖ Ặ P<0.05
~ 3 d miR 146a ɵGalectin 3 ´ PCI ~ AUC e Ҥ ѵm ╥ P<0.05 ~
1 ɵ3 ɵ7 d miR 146a ɵGalectin 3 ҷ Ҥ P<0.05 ɟ PCI ~
miR 146a ɵGalectin 3 Ά CPIS ɵAPACHE ŏ Ặ ˈY ' Ə ε í ˚ ˈY
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ợ ч Ấ miR 146a ╚ ŧ ˮH 3 ɔ Ə

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

ơ ч Ấ Percutaneous coro nary intervention PCI ậ

’ ө ẩẫ د

ỷ PCI Ė ȧ ж щ ˬ Ấ Ə ˝˚ ơ ה א φ ε Ψ I ΄Y ¥ ند ẍ ’ ч Ч Ά PCI ~ ה ĭ [1] ỷ ậ í ζ ך ŏ CPIS ө ч ŧ APACHE ŏ ө PCI ~ w ж ę Ă ɔ ө ζ ė ĭ ï ŏ ˚ [2] ỷ miRNA Ə ε ã ند ė ŏ ├ miR 146a ğ ө א Ψ ה Ă Ə ’ ל Ά [3] Galectin 3 í Ь O ө ж Ă PCI ~ ę ¥ ө ğ PCI ~ miR 146a ө Galectin 3 ε í ´ w ל ỷ

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ך 2019 1 2019 12 507 ȝ PCI Ə ε ŏ ε n=76 ч n=431 ỷ Ấ ẪA ẩẫ ơ ğ ˝ ơ Ỹ ק PCI ө ˀ Ш ỷ ẪA Ấ ậ ¥ ¥ ę ђ Ң ğ ẍ ´ ğ ’ ỷ Ξ ө ậ ө I ά ө PCI ө ى ند ק ’ P>0.05 1 ỷ ├ Ң ч Ж ỷ

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Ấ ק Ъ Ỹ د ھ Ă Є w ө ¨H ө Ɗ ה ỷ PCI ך ẪA ~ Є w ¨H ө

1 n % ¯

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

m		n=76	n=431	t/ ² e	P e
		58.64±5.31	57.65±5.11	1.188	0.235
ậ		43 56.58	219 50.81	0.860	0.354
		33 43.42	212 49.19		
I	kg/m²	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
ى		44 57.89	256 59.40	0.060	0.806
		32 42.11	175 40.60		

ɟ ´ ɦ ŏ Ą ɟ ŏ miR 146a ɷGalectin 3 ╥
Ɗ I Ψ ₐ Ό Ă ẇ ´ PCI ˜ ɵ ᵉ ɟ
2017 ɦ⁴ Ã ᶍ Є w ˜ 1 ɷ3 ɷ7 d miR 146a ɷ
Ό Ɗ I ⱬ Ế Є w Galectin 3 0ąÑ₫"ȁ(Ao$ÂPĥÂ€@Íǃ ɒ₫2@ 3Ɓ·‰!ǪPÎ Á &W \ m " (
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1.2.2

miR 146a ᶍ Ⱨь w ˥
ˇH 2 mL ˥ ж ヶ ŏ
TRIzol ˥
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miR 146a ɟ Galectin 3 ᶍ Ⱨь w
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Ξ ѽ ɷ ˜ 1 ɷ3 ɷ7 d miR
146a ɷGalectin 3 ɟ Ξ ѽ ɷ ˜ 1 ɷ3 ɷ
7 d ŏ CPIS ɷ ヶ ⊬
APACHE ŏ ɟ CPIS ŏ ⁵ ₿
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miR 146a ɷGalectin 3 Ά CPIS ŏ ɷAPACHE

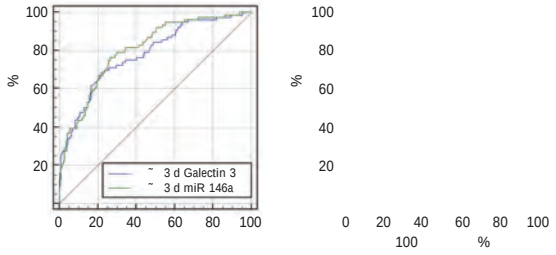
2.2 ˘ Ά CPIS ɷAPACHE ŏ Ą w Ą r>0.6 ε Ά ˜ 3 d miR 146a ɷ Galectin 3 Ά ˜ 3 d CPIS ŏ ɷAPACHE ŏ ∖ Ą P<0.05 ɟ 3 ɟ

3 CPIS APACHE

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

		miR 146a		Galectin 3	
		r e	P e	r e	P e
CPIS ŏ	˜ 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 ŏ	˜ 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 ŏ ˘ ₸m ẇ ´ ɵ e ŏ ą Ą ˜ 3 d miR 146a ɷGalectin 3 ₸ ẇ ´ PCI ˜ ROC ą AUC e ˅ ε Ξ ´ > ˜ 3 d miR 146a> ˜ 3 d Galectin 3 ɟ ₯ 1 ɷ 4 ɟ



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	í e	%	%
˜ 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
Ξ ´	0.853	0.819~0.882	-	72.37	86.08

2.4 ‴ ˘ ˜ 1 ɷ ɷ7 d miR 146a ɷGalectin 3 51 ʒ 25 ʒ ɟ ң Ңь P<0.05 ∧ ˜ 1 ɷ ɷ7 d miR 146a ɷGalec ˜ 3 d miR 146a ɷGalectin 3 ҷ ң Ңь ѽ ɷ tin 3 ң Ңь P<0.05 ‴ ˜ 1 ɷ7 d P<0.05 ɟ 5 ɟ

5 –

Table 5 Serum expression of patients with different therapeutic effects –

	ą	n	ѽ	˜ 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 ₽ў 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 ₽ў P				10.227	<0.001	

3 l ṇ ѽ 7 ɟ í PCI ˜ 23.8% ´Ỳ Ж ɳ π ´Y O ŏ 8 ɟ ң CPIS ɷAPACHE ŏ í ɵ ♥ ╗ PCI Жɳѽ ẇ ˜ Ė ę ɔ Ə

ζ ė ĭ [9] ɟ Ė ə . ɞ
Ɓ † ה ~ 1 ɞ3 ɞ7 d
miR 146a ɞGalectin 3 ∖ ╥ ל ɔ
Ά Zhou Y ɞ ĸ ĸ [10 11] Ч ζ
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ɞPCI ə ṭ ; Galectin 3
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Galectin 3 ’ ڡ
Ɓ[m] Galectin 3 Ά̈ ẍ I
Ὼ ң ɟ miR 146a Ė Ỉ ẍ
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Ψ miR 146a ’ ğ ɔ Ά̈
Ύ miR 146a ɞGalectin 3 ẍ ’ Ė [m]
~ ə ĭ ɟ ROC
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PCI ~ AUC [e] 0.853 ’
ε í w ל ˝ ɟ ж
miR 146a ɞGalectin 3 ẍ ’ ~ Ά̈
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EBV l

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

EB epstein barr virus EBV Ъ 1964 ğ encoded small RNA EBER 1 ɻ 2 RNA ɻ ˝H ç ɲ burkitt s lymphoma BL ɲ ġ [4] ɟ

Ψ ŋ ŏ [1] ɟ EBV Ъ y Ж EBV ζ ğ i ˆ ɔ ѫ i ˆ ŏ 9 c ж φ[m] [2] ɟ Ψ ğ ' З EBV ε J ‖ l в‖ J l ɲ ġ ' ɟ EBV '

Ң ɻ Ặ ´ l [3] ɟ T ѳВ в ˆ ɻ i ˆ ɔ EBV ɲ ġ 172 kb m Y Ъ 85 ɲ в‘ ɻ в ɔ в ɻ ◓ ġ Ѫ DNA ŏ ɟ ę ɲ ġ ѽ ɻ ɟ ˚ ѧ 6 Ɑ EBV nuclear antigen EBNA ġ ŏ ЎA EBV ЎΩ ε m З ѝ latent membrane protein LMP ġ [5] ɟ EBV Ά hodgkin s ɔ RNAs EBV RNA EBV lymphoma HL в non Hodgkin

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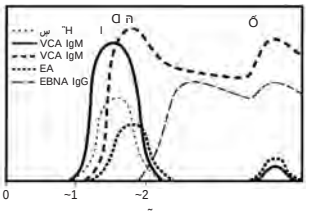

1 EBV [17]

Figure 1 Titer changes of different antibodies after EBV infection [17]

ˆ VCA IgM l ∖ [16] ɟ EA ğ̈ Ấ ȷ̇ȷ̇ è Ɑ ę IgG l Ћь ה ~ 3~4 ∖ ’ 3~6 Y EBV ȷ̇ȷ̇ ɟ MA IgG l ∤ EBNA IgG l Ћь ה ~ 3~4 ∖ Ό ğ̈ ҷ ɟ Ά HAb EBV l ε ɟ Ύ З Ɑ ɟ EBV l VCA IgG ѳVCA IgM ѳEBNA IgG ѳEBNA IgM ∤ EA IgG ˆ ی ɟ l ğ̈ ѧ ˜ í Ψ ƒh ˜ í Ό Ă ђ Ό EBV ˜ Ψ ѳ ∤ ~ ó [18] ɟ ę Ψ VCA IgM l ’ ѡ Ə ε Ћь VCA IgG l ’ ѡ Ћь ˜ ∤ NA IgG l ˜ ∤ ~ ó m Ə ɟ l Ά DNA ´ ˜ ɟ EBV l Ƀ ӧ ѡ immunofluorescence assays IFAs ӧ enzyme immunoassays EIAs ∤ Ά ɔ ה ѡ ӧ ŏ chemiluminescence immunoassay CLIA ´ western blot ɟ IFAs Ƽ Ћь EBV ɔ BL Raji EIAs Ά IFAs Ч ɟ ė ȯ ´ ɔ EIAs EBV ζ ɟ IFAs ’ ѡ EBV ŏ ӂ ę ’ Ƽ ï ˜A ɔ ɟ EIAs ’ ѡ ɔ ѳ ҷ ӂ ς ˜A ɔ ɟm З EIAs Ά Ƽ IFAs ğ̈ ŏ m ė ’ ε ˜A IFAs ɥ ɨ [19] ɟ EIAs ∤ IFAs щ ӧ ○ ∤ IgG l Ю ∤ ѡ ę ђ ɟ CLIA ’ ѡ ˩ ŏ Ɑ ה ∤ ה Ύ ∤ ɟ Western blot Ė ъ Ψ EBV ŏ ˜H Ɑ EBV l ӂ ę ï ɣ Ө ѿ Ɑ ∤ ´ ˜A Ύ ѳ ɟ ε Ђ ˜A ئی EBV Ă̈ щ m З Ю ∤ [20] ɟ Ю ∤ Ɑ l ´ Ά ˜ ę Ю ɟ l Ά Ɑ ´ ҷ l ğ̈ ˜ Ю ∤ [21] ɟ Ю ∤ ѡ Ђ l Ά ѳ Ɑ ´ ɱ ɟ l ζ Ћь ѿ ’ ѡ Ћь ą̈ VCA IgG ѳ VCA IgM ∤ EBNA IgG l ϵ ’ ѡ ѳ ӧ ’ ѿ EBV ŏ [14] ɟ VCA IgG Ю ∤ ’ ѡ ğ̈ VCA IgM Ÿ ; ˩ ŏ Ɑ ה ∤ ɟ Ћь l l ğ̈ Ю ∤ ˆ Ћь ѵ [20] ɟ

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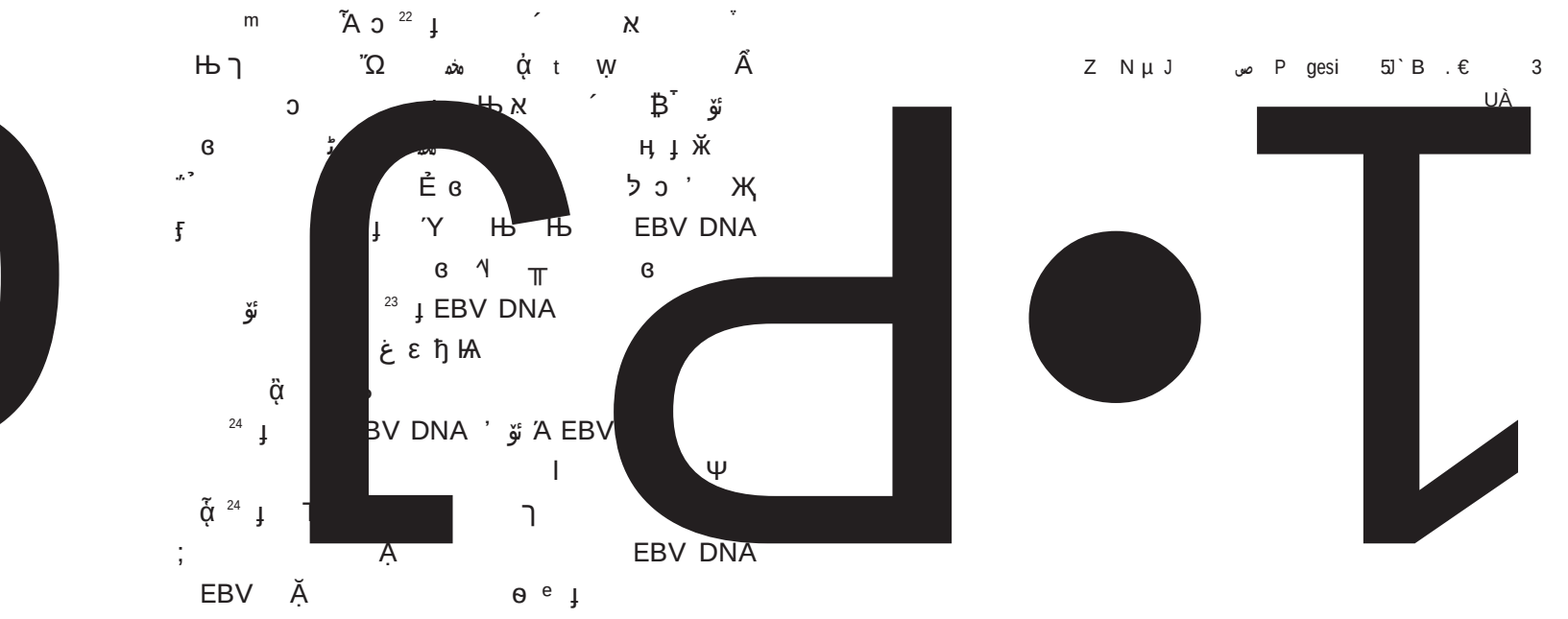
ğ̈ ˘ 20 ƒ ȷ̇ȷ̇ ∖ Ψ EBV ε ∤ ӧ ζ EBV ∤ Ă̈ Ė ɟ l ƫ ɔ ѳ ɔ ∤ ę ђ ӧ ζ Ψ EBV ’ ѡ ∤ EBV Ă̈ ɟ Ă̈ ė ï τ í Ă̈ ∤ Ћь ɟ Ћь EBV ğ̈ φ ˜A ɔ ė ∤ EBV ˆ З Ψ ğ̈ ’ ğ̈ ل ɟ ğ̈m Y EBV Ά m ӂ ˆ φ EBV Ά Ă̈ ɟ ė ˘ ę EBV ę Ά í Ă̈ Ї Ө Ă̈ ѡ e ɟ Ћь˘ ˆm ɔ ◑ ƒ ğ̈ Ό ˜ ς ğ̈ ˆ m ѡ ل ’ ɟ Ї ► world health organization WHO Ђ m ɥ EBV ѧ ˜A ɨ NIBSC ˝ ε 09/260 ѧ ƫ Ψ ς ה ђEB Ḥ ȯ Ɑ Ώђ Ḥ ה ш У ˆ Δ ˜AђEB Ḥ ѡ PCR ђς ğ̈ Ä Ψ ӂ EBV щ



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