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CORRELATION ANALYSIS OF SERUM FCAS1, PROCALCITONIN AND SCYA25 LEVELS WITH THE PROGNOSIS OF PATIENTS WITH SEPSIS

KORELACIONA ANALIZA NIVOA SERUMSKIH FCAS1, PROKALCITONINA I SCYA25 SA PROGNOZOM PACIJENATA SA SEPSOM

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Summary

Background: To explore the levels of serum Familial Cold Autoinflammatory Syndrome 1 (FCAS1), Procalcitonin, and Small Inducible Cytokine A25 (SCYA25) in patients with sepsis caused by drug-resistant *Klebsiella pneumoniae* (KP) infection and their relationship with prognosis.
Methods: The study group consisted of 410 patients who were hospitalised to our hospital between March 2019 and March 2024 and had sepsis brought on by KP. A total of 196 drug-resistant patients with sepsis caused by KPs were classified into the drug-resistant group, and 214 nondrug-resistant patients with sepsis caused by KPs were classified into the nondrug-resistant group. According to the severity of sepsis, 410 patients with KP-induced sepsis were divided into a severe sepsis group (178 patients) and a sepsis group (232 patients). The control group consisted of an additional 410 healthy volunteers who were physically examined at our hospital during the same time period. It was established that drug-resistant KPs were resistant to clinical medications. The prognosis of patients with drug-resistant sepsis caused by a KP infection was split into a survival group and a death group 28 days after admission. Serum FCAS1 levels were detected via an enzyme-linked immunosorbent assay, and serum Procalcitonin and SCYA25 levels were determined via the chemiluminescence method. The diagnostic value of serum

Kratak sadržaj

Uvod: Cilj je bio da se istraže nivoi serumskog proteina FCAS1 (Familial Cold Autoinflammatory Syndrome 1), prokalcitonina i malog indukovanog citokina A25 (SCYA25) kod pacijenata sa sepsom izazvanom infekcijom multirezistentnom bakterijom *Klebsiella pneumoniae* (KP), kao i njihovu povezanost sa prognozom bolesti.
Metode: U studiju je uključeno 410 pacijenata hospitalizovanih u našoj ustanovi od marta 2019. do marta 2024. godine, kod kojih je sepsa bila uzrokovana KP. Ukupno 196 pacijenata sa sepsom izazvanom multirezistentnom KP svrstano je u grupu rezistentnih, dok je 214 pacijenata sa sepsom izazvanom nerazistentnom KP svrstano u nerezistentnu grupu. Prema težini sepse, svih 410 pacijenata je podeljeno na grupu teške sepse (178 pacijenata) i grupu sepse (232 pacijenata). Kontrolnu grupu činilo je dodatnih 410 zdravih dobrovoljaca koji su u istom periodu obavili sistematski pregled u našoj ustanovi. Multirezistentnost KP je definisana kao rezistencija na klinički relevantne antimikrobne lekove. Prognoza pacijenata sa sepsom izazvanom multirezistentnim KP procenjena je 28 dana nakon prijema, čime su formirane grupa preživelih i grupa umrlih. Nivoi serumskog FCAS1 određeni su ELISA metodom, dok su nivoi prokalcitonina i SCYA25 određeni hemiluminiscencijom. Dijagnostička vrednost ovih parametara za

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FCAS1, Procalcitonin, and SCYA25 for drug-resistant KP-induced sepsis, as well as their predictive value for the mortality of patients with drug-resistant KP-induced sepsis, were examined using receiver operating characteristic (ROC) curves. Multivariate logistic regression analysis was used to investigate the factors impacting death in patients with sepsis and drug-resistant KP infection.

Results: A total of 250 drug-resistant KP strains were detected in 196 patients with sepsis caused by drug-resistant KPs. The resistance rates of these strains to cefazoline, ceftriaxone, ceftazidime, amtronam, imipenem and meropenem were greater than 90%. Serum FCAS1, Procalcitonin, and SCYA25 levels exhibited a statistically significant difference ($P<0.05$) between the severe sepsis group and the control and sepsis groups. Additionally, the sepsis group's levels were higher than those of the control group. Serum FCAS1, Procalcitonin, and SCYA25 levels were considerably higher in the drug-resistant group than in the nondrug-resistant group ($P<0.05$). The nonsurviving group had higher levels of serum FCAS1, Procalcitonin, SCYA25, Acute Physiology and Chronic Health Evaluation II (APACHE II), and infection-related Organ Failure Assessment (SOFA) ratings than the surviving group, while the nonsurviving group's serum ALB level was lower ($P<0.05$). Patients with drug-resistant KP infection and sepsis had higher serum levels of FCAS1, Procalcitonin, and SCYA25, which were risk factors for death, according to multivariate logistic regression analysis ($P<0.05$), whereas elevated levels of serum ALB were protective factors for death in patients with drug-resistant KP infection and sepsis ($P<0.05$). The results of the ROC curve analysis revealed that the areas under the curve (AUCs) of serum FCAS1, Procalcitonin, and SCYA25 and the combined diagnosis of drug-resistant KP infection sepsis were greater than those of FCAS1, Procalcitonin and SCYA25 ($P<0.05$), and the AUCs of the combined prediction of the three indicators for the death of patients with drug-resistant KP infection sepsis were greater than those of FCAS1, Procalcitonin and SCYA25. The AUC was calculated separately ($P<0.05$).

Conclusions: The levels of serum FCAS1, Procalcitonin and SCYA25 in patients with drug-resistant KP-induced sepsis are relatively high. The combined detection of these three indicators has high diagnostic value for drug-resistant KP-induced sepsis and is related to the death of patients.

Keywords: sepsis, drug resistance, *Klebsiella pneumonia*, nucleotide-binding oligomerisation domain-like receptor protein 3, Procalcitonin, small inducible cytokine A25

Rezultati: Kod 196 pacijenata sa sepsom izazvanom multirezistentnom KP izolovano je ukupno 250 rezistentnih sojeva. Stope rezistencije na cefazolin, ceftriakson, ceftazidim, amikacin, imipenem i meropenem bile su veće od 90%. Postojala je statistički značajna razlika u nivoima serumskog FCAS1, prokalcitonina i SCYA25 između grupe teške sepse i kontrolne i sepsa grupe ($P<0,05$), pri čemu su nivoi bili najviši u grupi teške sepse. Nivoi svih ispitivanih markera bili su značajno viši u rezistentnoj nego u nerezistentnoj grupi ($P<0,05$). Grupa umrlih imala je više nivoe FCAS1, prokalcitonina, SCYA25, APACHE II i SOFA skorova, dok je nivo serumskog ALB bio niži ($P<0,05$). Logisticka regresija je pokazala da su povišeni nivoi FCAS1, prokalcitonina i SCYA25 faktori rizika za smrt, dok je viši nivo ALB bio protektivni faktor ($P<0,05$). ROC analiza pokazala je da kombinovana dijagnostika korišćenjem sva tri markera ima veću površinu ispod krive (AUC) od pojedinačnih markera ($P<0,05$), kako za dijagnostiku multirezistentne KP sepse, tako i za predikciju smrtnog ishoda.

Zaključak: Nivoi serumskog FCAS1, prokalcitonina i SCYA25 značajno su povišeni kod pacijenata sa sepsom izazvanom multirezistentnom *Klebsiella pneumoniae*. Kombinovano određivanje ovih biomarkera ima visoku dijagnostičku i prognostičku vrednost i povezano je sa smrtnim ishodom.

Ključne reči: sepsa, rezistencija na lekove, *Klebsiella pneumoniae*, NLRP3, prokalcitonin, mali indukovani citokin A25

Introduction

A typical consequence of trauma or injury, sepsis is a potentially fatal state of organ malfunction brought on by the host's dysregulated reaction to infection (1–3). *Klebsiella pneumoniae* (KP) is one of the main pathogenic bacteria that causes sepsis and has strong disseminated infectivity (4). Although most KP isolates are sensitive to antibacterial drugs, owing to the previous empirical and preventive overuse of broad-spectrum carbapenem antibacterials, the detection rate of highly virulent, strongly resistant isolates has been increasing annually. However, carbapenem-resistant highly virulent KPs are extremely resistant to ampicillin, imipenem, meropenem and ceftazidime. This not only reduces the choice of drugs for sepsis treatment but also increases the risk of treatment failure, thereby increasing the mortality rate of sepsis patients. Therefore, timely diagnosis and identification of drug-resistant KPs can improve the therapeutic effect in patients with sepsis (5). The multiprotein complex known as the Familial Cold Autoinflammatory Syndrome 1 (FCAS1) inflammasome is mainly expressed by monocytes and dendritic cells. It is composed of three proteins: the caspase-1 precursor, apoptosis-associated speck-like protein (ASC), and NOD-like receptor (NLR). It is a component of the innate immune system (6). It is closely related to cellular inflammation, autophagy, pyroptosis and apoptosis. Procalcitonin is a procalcitonin propeptide and an important proinflammatory

cytokine. Its level increases during inflammation or infection in the body and can be used as a biomarker for diagnosing infection (7–9). Small Inducible Cytokine A25 (SCYA25) is expressed mainly in the thymus and small intestinal epithelium (10). It is the sole ligand of C-C chemokine receptor 9 (CCR9), participates in various inflammatory diseases and promotes inflammatory responses (11–13).

There are relatively few studies (14–16) on the roles of FCAS1, Procalcitonin, and SCYA25 in drug-resistant KP-induced sepsis. Therefore, this study aimed to analyse changes in the levels of FCAS1, Procalcitonin, and SCYA25 in the serum of patients with drug-resistant KP infection sepsis and to explore their predictive value for the diagnosis and prognosis of this condition.

Materials and Methods

General information

The study group consisted of 410 patients with sepsis brought on by KP who were hospitalised to our hospital between March 2019 and March 2024. A total of 196 drug-resistant patients with sepsis caused by KPs were classified into the drug-resistant group, and 214 nondrug-resistant patients with sepsis caused by KPs were classified into the nondrug-resistant group. According to the severity of sepsis, 410 patients with KP-induced sepsis were divided into a severe sepsis group (178 patients) and a sepsis group (232 patients). A control group of 410 healthy volunteers who were examined during the same time period at our hospital was selected. The research group included 202 female patients and 208 male participants. With an average age of 50.35±8.78 years, the age range was 34 to 75 years. A total of 204 females and 206 males made up the control group. The average age was 51.62±8.32 years, with a range of 35 to 78 years. There was no statistically significant difference in sex or age between the study and control groups ($P>0.05$), and the groups were comparable.

Inclusion criteria: (1) Meeting the relevant diagnostic criteria for sepsis; (2) The blood culture results showed KP infection, and the patients in the drug-resistant group were resistant to antibacterial drugs such as cefazolin, ceftazidime, amtronam, levofloxacin, and imipenem. (3) Complete clinical data.

Exclusion criteria: (1) Combined with autoimmune deficiency diseases and malignant tumours; (2) Severe dysfunction of the liver and kidneys; (3) Women who are breastfeeding or pregnant; (4) Mental abnormalities and cognitive dysfunction.

This study was approved by the hospital's medical ethics committee (SYSU-FAH-2019-022), and all participants provided informed consent.

Data collection

The body mass index (BMI), history of drinking, smoking, high blood pressure, diabetes, and coronary heart disease, platelet count (PLT), white blood cell count (WBC), and neutrophil count (NEU), as well as the levels of albumin (ALB), alanine aminotransferase (ALT), aspartate aminotransferase (AST), and infection-related organs of patients with sepsis were recorded. The failure assessment (SOFA) score and Acute Physiology and Chronic Health Evaluation II (APACHE II) score were recorded.

Analysis of drug sensitivity tests for drug-resistant strains

When pathogenic bacteria are collected, the laboratory operation guidelines should be strictly followed. Bacterial identification and drug-sensitivity tests should be conducted using the Mérieux VITEK 2 fully automated microbial identification instrument, and the specific drug resistance of the strains should be determined by the microdilution method.

Detection of serum FCAS1, Procalcitonin and SCYA25 levels

On the day of admission for the study group and on the day of physical examination for the control group. The blood was centrifuged at 3,000 r/min for 15 minutes (with a centrifugal radius of 10 cm), and the upper serum layer was removed. Serum FCAS1 (Shanghai Ulink Life Science Co., LTD., item number: YLK-E1955) levels were detected via enzyme-linked immunosorbent assay (ELISA), and Procalcitonin (Beckman Coulter Inc., USA, item number: 339114) and SCYA25 (Shanghai Boke Biotechnology Co., LTD., item number:) levels were determined via the chemiluminescence method. The reagent kit's instructions should be strictly followed when performing the BKE8014 level test.

Laboratory testing methods

All serum samples were aseptically collected within 24 hours after the patient's admission. The serum was separated after centrifugation at 3000 rpm for 15 minutes, then aliquoted and stored at -80 °C for testing. Concentration determination of target biomarkers in serum was performed using enzyme-linked immunosorbent assay (ELISA).

(1) The concentration of human FCAS1 protein was quantitatively detected using the human FCAS1 ELISA Kit (item number: ml002453) produced by Shanghai Enzyme-Linked Biotechnology Co., LTD.

(2) The level of Procalcitonin was determined using the human procalcitonin detection kit (chemiluminescence method, item number: CL0312) from

Shenzhen Feipeng Biotechnology Co., LTD.

(3) The concentration of the chemokine SCYA25 was detected using the human SCYA25/TECK DuoSet ELISA Kit (item number: DY385) from R&D Systems of the United States.

All experimental operations were carried out strictly in accordance with the standard procedures specified in the instructions for their respective reagent kits, including sample dilution, sample addition, incubation, washing, colour development, and reaction termination. Ultimately, the absorbance values (OD values) were read at specific wavelengths using a microplate reader (such as BioTek Synergy H1), and the precise concentrations of FCAS1, Procalcitonin, and SCYA25 in each sample (in units of ng/mL or pg/mL, respectively) were calculated based on the standard curves provided by the kit. During the experiment, standard, quality control, and blank controls were used to ensure the accuracy and reliability of the test results.

Statistical processing methods

SPSS 25.0 was used to analyse the data. To represent measurement data with a normal distribution, use the notation $\bar{x}\pm s$. One-way analysis of variance was used for comparisons among multiple groups, the independent-samples t-test for comparisons between two groups, and the least significant difference (LSD) test for pairwise comparisons within multiple groups. Count data were expressed as counts or percentages, and the χ^2 test was used for comparisons between groups. Serum FCAS1, Procalcitonin, and SCYA25 were evaluated for their diagnostic value in drug-resistant KP-induced sepsis and their prognostic value for mortality in patients with this condition using receiver operating characteristic (ROC) curves. The factors influencing death in patients with drug-resistant KP infection and sepsis were examined using multivariate logistic regression analysis. A P value <0.05 was considered to indicate statistical significance.

Results

Drug resistance status of patients with sepsis caused by drug-resistant KPs

A total of 250 drug-resistant KP strains were detected in 196 patients with sepsis caused by drug-resistant KPs. The resistance rates of these strains to cefazoline, ceftriaxone, ceftazidime, amtronam, imipenem and meropenem were greater than 90%, see *Table I*.

Among the 196 patients with sepsis caused by drug-resistant Klebsiella pneumoniae (KP) infection included in this study, the analysis of bacterial drug resistance showed that the isolated strains exhibited high multidrug resistance. These drug-resistant strains have demonstrated widespread and significant resistance to a variety of commonly used clinical antibacterial drugs, including β-lactam types (such as cefzolin, ceftriaxone, ceftazidime, and amtronam) and carbapenems (such as imipenem and meropenem).

Comparison of serum FCAS1, Procalcitonin and SCYA25 levels among the control group, the sepsis group and the severe sepsis group

The sepsis group's levels were higher than those of the control group, and the difference was statistically significant ($P<0.05$) for serum FCAS1, Procalcitonin, and SCYA25 levels between individuals with severe sepsis and the control and sepsis groups (see *Table II*).

The levels of serum FCAS1, Procalcitonin, and SCYA25 in the control group, sepsis group, and severe sepsis group were compared. The results indicated that as sepsis severity increased, the serum levels of FCAS1, Procalcitonin, and SCYA25 significantly increased. Specifically, the levels of serum FCAS1, Procalcitonin and SCYA25 in the severe sepsis group were significantly higher than those in the sepsis group, and in turn, the levels in the sepsis group were higher than those in the healthy control group. These findings suggest that serum levels of FCAS1, Procalcitonin, and SCYA25 are closely associated with sepsis severity and may reflect it.

Table I Drug resistance status of Klebsiella pneumoniae in patients with sepsis [n(%)].

Antimicrobial agents	Drug resistance	Antimicrobial agents	Drug resistance
Cefazolin	250 (100.00)	Imipenem	236 (94.40)
Ceftriaxone	246 (98.40)	Melopeinan	230 (92.00)
Ceftazidime	248 (99.20)	Gentamicin	164 (65.60)
Aztreonam	242 (96.80)	Tigecycline	50 (20.00)
Levofloxacin	144 (57.60)	Etapenem	138 (55.20)

Table II Comparison of serum FCAS1, Procalcitonin, and SCYA25 levels between drug-resistant and non drug-resistant groups ($\bar{x}\pm s$).

Group	n	FCAS1 (pg/mL)	Procalcitonin (g/L)	CCI25 (ng/L)
Severe sepsis group	178	195.62±39.87	8.35±1.98	356.71±58.20
Sepsis group	232	137.55±20.71	5.27±1.12	280.52±40.21
Control group	410	50.39±10.52	1.67±0.21	107.55±19.69
t		1405.520	1179.560	1676.480
P		<0.001	<0.001	<0.001

Table III Comparison of serum FCAS1, Procalcitonin, and SCYA25 levels among the control group, sepsis group, and severe sepsis group ($\bar{x}\pm s$).

Group	n	FCAS1 (pg/mL)	Procalcitonin (μg/L)	SCYA25 (ng/L)
Drug resistance group	196	174.51±22.62	7.18±1.26	337.42±40.35
Nonresistant group	214	151.98±18.79	6.08±0.95	291.88±32.60
t		7.800	7.282	8.930
P		<0.001	<0.001	<0.001

Table IV Diagnostic value of serum FCAS1, Procalcitonin, SCYA25 for sepsis caused by drug-resistant KP infection.

Indicator	Sensitivity (%)	Specificity (%)	Optimal truncation value	AUC	AUC 95%CI	P	Yoden Index
FCAS1	78.50	73.86	161.70 pg/mL	0.821	0.775~0.887	<0.05	0.527
Procalcitonin	75.54	71.99	6.46 μg/L	0.815	0.757~0.873	<0.05	0.488
SCYA25	76.56	86.95	319.83 ng/L	0.824	0.765~0.884	<0.05	0.638
3 Joint Projects	89.83	95.36	–	0.917	0.865~0.969	<0.05	0.854

Comparison of serum FCAS1, Procalcitonin and SCYA25 levels between the drug-resistant group and the nondrug-resistant group

The levels of serum FCAS1, Procalcitonin, and SCYA25 in the drug-resistant group were significantly higher than those in the nondrug-resistant group (P<0.05; see *Table III*).

The levels of serum FCAS1, Procalcitonin, and SCYA25 were compared between the drug-resistant and non-drug-resistant groups. The results showed that the levels of these indicators in the drug-resistant group were significantly higher than those in the non-drug-resistant group. Drug resistance may be associated with increased levels of FCAS1, Procalcitonin, and SCYA25 in serum. Changes in these indicators may reflect the severity of drug-resistant Klebsiella pneumoniae (KP) infection and patients' immune responses. The elevated levels of serum FCAS1, Procalcitonin, and SCYA25 are closely associated with the presence of drug-resistant bacteria, suggesting that these indicators have potential clinical value for evaluating the clinical manifestations and prognosis of drug-resistant KP infections.

Diagnostic value of serum FCAS1, Procalcitonin and SCYA25 for sepsis caused by drug-resistant KP infection

ROC curve analysis was conducted with KP resistance in patients with sepsis (yes =1, no =0) as the state variable and serum FCAS1, Procalcitonin, and SCYA25 levels as the test variables. The area under the curve (AUC) values for serum FCAS1, Procalcitonin, and SCYA25 alone, and for the combination of the three indicators for diagnosing drug-resistant KP-induced sepsis, were 0.821, 0.815, 0.824, and 0.917, respectively. The AUC of the combined diagnosis of the three indicators was greater than that of the individual diagnoses of FCAS1, Procalcitonin, and SCYA25 (Z=3.780, 3.897, and 3.925, respectively; all P<0.05), as shown in *Table IV*.

Table V Comparison of clinical data between survival group and death group [n (%) or $\bar{x}\pm s$].

Item	Survival group (n=118)	Death group (n=78)	x^2/t	P
Gender			0.144	0.700
Male	62 (52.54)	44 (56.41)		
Female	56 (47.46)	34 (43.59)		
Age (years)	49.60±5.44	50.15±5.62	0.398	0.697
BMI (kg/m^2)	22.15±1.59	22.64±1.62	-1.475	0.147
Smoking history			0.085	0.777
Yes	64 (54.24)	40(51.28)		
No	54 (45.76)	38 (48.72)		
Drinking history			0.756	0.389
Yes	56 (47.46)	44 (56.41)		
No	62 (52,54)	34 (43.59)		
History of hypertension			0.414	0.524
Yes	68 (57.63)	50 (64.10)		
No	50 (42.37)	28 (35.90)		
History of diabetes			0.085	0.777
Yes	64 (54.24)	40 (51.28)		
No	54 (45.76)	38(48.72)		
History of coronary heart disease			0.903	0.346
Yes	52 (44.07)	42 (52.85)		
No	66 (55.93)	36 (46.15)		
PLT (×109/L)	117.59±15.37	121.27±16.51	-1.129	0.266
WBC (×109/L)	13.51±1.75	13.99±2.01	-0.987	0.320
NEU (×109/L)	8.75±1.18	9.10±1.29	-1.828	0.074
ALB (g/dL)	2.99±0.39	2.56±0.22	6.230	0.001
ALT (U/L)	43.51±5.39	44.15±5.81	-0.40	0.643
AST (U/L)	39.26±4.59	40.15±4.81	-0.95	0.363
SOFA rating (points)	9.85±1.30	11.9±1.52	6.039	<0.001
APACHEI score (points)	18.22±2.38	23.10±3.19	-8.751	<0.001
FCAS1 (pg/mL)	167.32±18.08	185.49±21.38	-4.501	<0.001
Procalcitonin (ng/L)	6.74±0.98	7.84±1.16	5.203	<0.001
CCI25 (ng/L)	328.54±20.35	351.01±24.74	-4.938	<0.001

Table VI Mortality from drug-resistant KP infection and sepsis in patients using multivariate logistic regression analysis.

Factor	β	SE	Wald χ^2	OR	OR95%CI	P
ALB	-0.303	0.128	5.754	0.744	0.583 0.940	0.019
FCAS1	0.851	0.328	6.976	2.351	1.241 4.463	0.001
Procalcitonin	0.589	0.255	5.392	1.799	1.099 2.946	0.023
SCYA25	1.137	0.469	5.911	3.100	1.249 7.748	0.018

Table VII The predictive value of serum FCAS1, Procalcitonin, and SCYA25 for mortality in patients with drug-resistant KP sepsis.

Indicator	Sensitivity (%)	Specificity (%)	Optimal truncation value	AUC	AUC 95%CI	P	Yoden Index
FCAS1	82.08	81.39	173.76 pg/mL	0.777	0.674 0.871	<0.05	0.637
Procalcitonin	84.65	86.47	7.41μg/L	0.821	0.736 0.926	<0.05	0.714
SCYA25	82.08	84.78	341.12 ng/L	0.832	0.758 0.926	<0.05	0.661
3 Joint Projects	84.65	91.56	-	0.939	0.887 0.980	<0.05	0.765

Comparing the survival group's clinical data with that of the nonsurviving group

Within 28 days, 78 out of 196 patients with drug-resistant KP infection sepsis died, with a case fatality rate of 39.83%. The percentages of sex, age, history of alcohol use, history of smoking, BMI, history of hypertension, history of coronary heart disease, history of diabetes, and PLT, WBC, NEU, ALT, or AST levels did not differ statistically significantly between the survival group and the nonsurviving group (P>0.05). While the nonsurviving group's serum FCAS1, Procalcitonin, and SCYA25 levels, as well as their APACHE II and SOFA scores, were considerably higher than those of the surviving group (P<0.05), the nonsurviving group's serum ALB level was lower than that of the surviving group (14, 16).

Multivariate logistic regression investigation of death rates in individuals with drug-resistant KP infection-induced sepsis

Stepwise regression was used to eliminate APACHE II and SOFA scores from the dependent variable, which was the prognosis of patients with drug-resistant KP infection and sepsis (survival = 0, death = 1). Multivariate logistic regression was conducted with serum ALB, FCAS1, Procalcitonin, and SCYA25 (original values entered) as independent variables. The results revealed that elevated levels of serum FCAS1, Procalcitonin, and SCYA25 were risk factors for death in patients with drug-resistant KP infection and sepsis (P<0.05), whereas elevated levels of serum ALB were protective factors for death in patients with drug-resistant KP infection and sepsis (P<0.05), see *Table VI*.

Predictive value of serum FCAS1, Procalcitonin and SCYA25 for the death of patients with drug-resistant KP infection and sepsis

ROC curve analysis was conducted with the prognosis of patients with drug-resistant KP infection sepsis (survival = 0, death = 1) as the state variable and serum FCAS1, Procalcitonin, and SCYA25 levels as the test variables. The results revealed that the AUCs of serum FCAS1, Procalcitonin, and SCYA25 alone, and of the combination of the three indicators, for predicting death in sepsis patients with drug-resistant KP infection were 0.777, 0.821, 0.832, and 0.939, respectively. The AUC of the combination of the three indicators for predicting the death of patients with drug-resistant KP infection in sepsis patients was greater than that of FCAS1, Procalcitonin, and SCYA25 alone in patients with UC (Z=0.934, 2.095, 2.334; all P<0.05), see *Table VII*.

Discussion

Sepsis is a severe systemic inflammatory response syndrome and is the leading cause of death among infected patients worldwide (17). KP is a common pathogen of sepsis. In recent years, owing to the extensive use of third-generation cephalosporins, the resistance of patients with KP-infected sepsis to various antibacterial drugs, such as ampicillin, ceftazidime, and ceftriaxone, has been increasing, mak-

ing the treatment of drug-resistant KP-infected sepsis more difficult (18–20). Therefore, timely identification of drug-resistant KP-induced sepsis is essential for patient treatment and prognosis (21).

Both the sepsis and control groups in this study had lower serum FCAS1 levels than the severe sepsis group, whereas the sepsis group had higher levels than the control group. Their serum FCAS1 levels may indicate the severity of their sepsis. Furthermore, the drug-resistant group had a higher blood FCAS1 level than the nondrug-resistant group ($P<0.05$). FCAS1 is a cytoplasmic immunological factor that responds to signals of cellular stress and participates in biological processes such as inflammation, immunity, and metabolism (22). It is typically triggered by an inflammatory response or host infection, which encourages apoptosis and the release of proinflammatory cytokines. It is closely related to the occurrence and development of sepsis. FCAS1 plays a significant role in infection (23). Research (24–26) has shown that activated FCAS1 can trigger apoptosis and cause inflammatory responses. When multidrug-resistant bacteria cause severe pneumonia, the FCAS1 signalling pathway is activated, inducing the massive production of the proinflammatory cytokine interleukin (IL)-1β and triggering an inflammatory cascade. An elevated serum level in patients with severe pneumonia complicated by multidrug-resistant bacterial infection is helpful for diagnosing this condition (27).

While the sepsis group had higher serum Procalcitonin levels than the control group, both groups had lower levels than the severe sepsis group (28). Patients with sepsis may have a serum Procalcitonin level that indicates the severity of their illness. The drug-resistant group had higher serum procalcitonin levels than the nondrug-resistant group ($P<0.05$). The hormone precursor of calcitonin, Procalcitonin, is secreted by thyroid C cells. Under normal physiological conditions, the serum Procalcitonin level can be ignored (29). However, during infection, its level increases rapidly, and it is a serological indicator for the clinical diagnosis of sepsis. Studies have shown that when the body is infected with gram-negative bacteria, under the action of cellular immunity, gram-negative bacteria undergo large-scale lysis and death, releasing a large amount of endotoxin. Endotoxins can increase serum Procalcitonin levels, aggravate the inflammatory response, and easily lead to a poor prognosis. Therefore, the serum Procalcitonin level can be used as a biomarker for predicting the prognosis of patients with sepsis caused by pandrug-resistant gram-negative bacteria (30). Procalcitonin is produced in significant amounts during an inflammatory response or a serious illness, which increases levels of inflammatory factors. Consequently, sepsis patients have elevated serum Procalcitonin levels, which correlate with the severity of the patient's illness and prognosis (31).

The group with severe sepsis had a higher serum SCYA25 level than both the sepsis and control groups, and the sepsis group had a higher level than the control group. The variations were statistically significant ($P<0.05$), indicating that, in sepsis patients, the serum SCYA25 level may be a good indicator of illness severity (32). Additionally, the drug-resistant group's serum SCYA25 level was higher than the nondrug-resistant group's. This may be because SCYA25 regulates inflammatory responses, white blood cell migration, and immunological differentiation, in addition to controlling a variety of immune cells. Studies (33–35) have shown that serum SCYA25 levels are higher in patients with sepsis than in healthy controls. The overexpression of SCYA25 can reduce the level of endothelial barrier tight junction proteins and increase the level of the proinflammatory cytokine nuclear transcription factor B (NF- B). Inhibition of SCYA25 can significantly inhibit the expression of proinflammatory cytokines. Another study reported that serum NF- B levels are elevated in patients with severe pneumonia caused by multidrug-resistant infections, suggesting that inhibiting NF- B-mediated inflammation can reduce the incidence of multidrug-resistant infections in these patients. SCYA25 can activate Toll-like receptor 4, promote the phosphorylation of P38, and promote the expression of various proinflammatory cytokines, such as IL-6. Its level increases in the serum of patients with acute lung injury secondary to sepsis; it also influences the likelihood of acute lung damage (36).

In this study, a total of 125 drug-resistant KP strains were detected in 98 patients with sepsis caused by drug-resistant KPs. The resistance rates to cephalosporin drugs (cefazolin, ceftriaxone, and ceftazidime), amtronam, imipenem, and meropenem were greater than 90%, whereas the resistance rates to levofloxacin, gentamicin, tegamycin, and ertapenem were relatively low. When treating patients with drug-resistant KP infection-induced sepsis, specific drug resistance should be detected, and symptomatic treatment should be carried out to improve the therapeutic effect. The results of the ROC curve analysis indicated that the AUC of the combined diagnosis of drug-resistant KP infection-induced sepsis by FCAS1, Procalcitonin, and SCYA25 was 0.917, which was greater than the AUC of the individual diagnosis of FCAS1, Procalcitonin, and SCYA25 ($P<0.05$), suggesting that serum FCAS1, Procalcitonin, and SCYA25 could be used for the diagnosis of drug-resistant KP infection-induced sepsis. The combined detection of these three indicators has increased diagnostic efficiency and can play an important role in the clinical auxiliary diagnosis of drug-resistant KP-induced sepsis. Within 28 days of admission, 78 out of 196 patients with drug-resistant KP-induced sepsis died, with a case fatality rate of 39.83%. Elevated serum ALB levels are a protective factor against death in patients with drug-resistant KP infection and sepsis

($P<0.05$), suggesting that, in clinical practice, changes in serum ALB, FCAS1, Procalcitonin, and SCYA25 levels in these patients should be closely monitored. If any abnormalities are found, timely treatment should be provided to improve prognosis. According to the study's ROC curve analysis, the combined prediction of death by serum FCAS1, Procalcitonin, and SCYA25 in patients with drug-resistant KP infection with sepsis had an AUC of 0.939. This might be because FCAS1, Procalcitonin and SCYA25 can promote the expression of proinflammatory cytokines, and the three indicators are interrelated. The combined effect plays a role in the prognosis of patients with drug-resistant KP infection-induced sepsis; thus, the combined predictive efficacy is greater. These findings further indicate that the serum levels of FCAS1, Procalcitonin, and SCYA25 can be used as biomarkers for predicting the death of patients with drug-resistant KP-induced sepsis. Moreover, these data can be combined with the general information of patients to assess the prognosis of patients with drug-resistant KP-induced sepsis on time and provide timely treatment. These findings could be used in the clinical diagnosis and treatment of drug-resistant KP-induced sepsis.

Conclusion

The combination of these three factors can significantly enhance the predictive efficacy of prognosis in patients with drug-resistant KP infection-induced sepsis. However, the sample size of this study was small, and some biases may have occurred during data analysis, affecting the accuracy of the results. Consequently, multicenter sampling and larger sample sizes are still needed to improve the accuracy and generalizability of the results. Moreover, the specific mechanisms by which FCAS1, Procalcitonin, and SCYA25 influence the prognosis of patients with drug-resistant KP-induced sepsis remain unclear. Further in-depth research and verification are needed to improve the diagnosis and prognosis of drug-resistant sepsis caused by KP infection.

Conflict of interest statement

All the authors declare that they have no conflict of interest in this work.

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