Prognostic value of serum amyloid A in patients with COVID-19

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Research Article

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Abstract

Objective: To investigate the prognostic value of serum amyloid A (SAA) in the patients with Corona Virus Disease 2019(COVID-19).

Methods The medical data of 89 COVID-19 patients admitted to Renmin Hospital of Wuhan University from January 3, 2020 to February 26, 2020 were collected. 89 cases were divided into survival group (53 cases) and non-survival group (36 cases) according to the results of 28-day follow-up. The SAA levels of all patients were recorded and compared on 1day after admission (before treatment) and 3day, 5day, 7day after treatment. The *ROC* curve was drawn to analyze the prognosis of patients with COVID-19 by SAA.

Results: The difference of comparison of SAA between survival group and non-survival group before treatment was not statistically significant, Z^1 = -1.426, P = 0.154. The Z^1 values of the two groups of patients at 3d, 5d, 7d after treatment were -5.569, -6.967, and -7.542, respectively. The P values were all less than 0.001, and the difference was statistically significant. The *ROC* curve results showed that SAA has higher sensitivity to prognostic value for death, the *AUC* area of 3d, 5d, 7d, which were 80.6%, 97.2%, 86.1%, and 96.1%, respectively.

Conclusion: SAA can be used as a death predictor of the prognosis in patients with COVID-19.

1 Introduction

Since December 2019, cases of pneumonia of unknown origin has been reported in Wuhan, Hubei Province. On January 7, 2020,the virus was identified as severe acute respiratory syndrome coronavirus 2(SARS-CoV–2)[1].The World Health Organization named it COVID–19.Until 5pm on March 10,2020,a total of 113,702 COVID–19 cases have been diagnosed and 4012 deaths all over the world, affecting 110 countries [2–3], with a fatality rate of 3.53%, and a higher mortality rate among older men with severe underlying diseases. [4]. It is important for those patients with COVID–19 to judge the prognosis of patients accurately, and to take more active and effective treatment measures. Currently SAA is commonly used as one of the indicators of inflammation monitoring in clinical practice [5–10]. This article analyzes the dynamic changes of SAA in 89 patients with COVID–19 in Renmin Hospital of Wuhan University to evaluate the clinical prognostic value of SAA, which is reported as follows.

2 Methods

2.1 Research objective

The data of 89 patients with COVID-19 who were admitted to Renmin Hospital of Wuhan University from January 3, 2020 to February 26, 2020 were collected, including 49 males and 40 females, aged 21-96 years, mean age (59.74±16.42) years old; 26 severe cases and 63 critical cases among total 89 patients, no mild and common cases were included. according to□Guidance for Corona Virus Disease 2019

Prevention, Control, Diagnosis and Management[11].All cases follow-up visited, according the 28-day follow-up results. And the follow-up date was March. 26.2020.

Inclusion criteria [11]: Those who meet one of the following criteria for the diagnosis of severe cases: ① Respiratory distress, RR≥30 breaths/min; ② Pulse oxygen saturation (SpO2)≤93% on room air at rest state; ③ Arterial partial pressure of oxygen (PaO2) / oxygen concentration (FiO2)≤300 mmHg (1 mmHg=0.133 kPa); ④ Patients with >50% lesions progression within 24 to 48 hours in pulmonary imaging; Those who meet one of the following criteria are diagnosed as critically ill cases: ① Respiratory failure occurs and mechanical ventilation is required; ② Shock occurs; ③ Complicated with other organ failure that requires monitoring and treatment.

Exclusion criteria: ① age <18 years; ② pregnant or lactating women; ③ treatment period less than 7 days; ④ patients with end-stage liver and kidney failure, advanced malignant tumors and other serious underlying diseases; ⑤patients and their families who signed DNR beforehand; ⑥ Patients who lost contact during follow-up.

2.2 Research method

2.2.1 Materials and Grouping method

Collect general information, physical examination, and supporting information of all patients within 6 hours after admission, collect blood samples of patients within 24 hours including blood routine, blood biochemistry, coagulation function, D-dimer, et al. Indicators including the SAA results of all patients were recorded on the 1d of admission (before treatment), 3d, 5d, and 7d after treatment, and to follow-up we contacted all patients by telephone after 28d. Based on the 28-day follow-up results, we divided 89 cases with COVID-19 into survival groups (53 cases) and non-survival group (36 patients).

2.2.2 Monitoring indicators and detection methods

Patients in both groups were treated according to the guidelines [11], including conventional treatment including early effective oxygen therapy, glucocorticoids, antiviral and nutritional support. 5ml of venous blood was drawn by fasting in the early morning to measure the SAA level on the 1d(before treatment), 3d, 5d, and 7d after treatment.

After the venous blood was collected, it was centrifuged at 4℃(speed of rotation: 3000r / min x 3min,and the centrifuge radius was 22cm) .The upper serum was taken and stored in a refrigerator at -20℃for testing. SAA was detected by immunoturbidimetry (Hitachi 7170 automatic biochemical instrument), and SAA normal value <10mg/L. All parameter settings and experimental steps are carried out in strict accordance with its operating procedures.

2.3 Statistical methods

After data collection, SPSS 26.0 software was used to analyze the data. The count data was expressed as[cases(%)], and comparison between groups was performed using c^2-test. Measurement data were expressed as mean ± standard deviation *( ),*and comparison between groups was performed using the independent sample *t*-test. The two groups of patients used SAA on the 1d (before treatment), and 3d, 5d, 7d after treatment were compared using rank sum tests. The *ROC* curve was used to evaluate the predictive value of SAA at the 1d, 3d, 5d, and 7d levels. The Z^2 value was used for comparison between groups. P <0.05 was considered statistically significant.

3 Results

3.1 Comparison of baseline data between survival and non-survival groups

As shown in the baseline data in Table 1, the non-survival group was older than the survival group (P<0.001); the non-survival group the patients number with dyspnea symptoms and decreased blood oxygen saturation was more than the survival group (P= 0.043, P<0.001); the non-survival group patients number with hypertension and cluster-onset was more than those in the survival group (P <0.001, P= 0.014); there were no statistically significant differences in gender composition ratio, heart rate, blood pressure, respiratory frequency, body temperature and other symptoms and previous medical history in the two groups.

3.2 Comparison of indicators before treatment in survival group and non-survival group

As shown in Table 2, the leukocyte count, neutrophil count, procalcitonin, lactate dehydrogenase, urea nitrogen, creatinine, blood glucose and D-dimer in the death group were higher than those in the survival group, all P<0.05, The difference was statistically significant; the lymphocyte counts, hemoglobin, albumin, and antithrombin III activity values in the non-survival group were all lower than those in the survival group, all P <0.05, and the difference was statistically significant

3.3 Comparison of SAA before and after treatment in survival group and non-survival group

With the progress of treatment, the Z^1-value of SAA before treatment in patients in the survival group and the non-survival group was -1.426, P= 0.154, and the difference was not statistically significant. The Z^1value of SAA was compared between the two groups at 3d, 5d, and 7d after treatment which was Respectively -5.569, -6.967, -7.542, P<0.001, with statistical significance (Figure 1). It is suggested that the difference of SAA levels between the two groups becomes larger and larger with the extension of treatment time. The significant increases in SAA levels indicates that the patient is at higher risk of eventual death.

3.4 The predictive value of SAA in the outcome of COVID-19 patients

According to the analysis results of the *ROC* curve, the *AUC* of SAA at each time point are: 0.588, 0.848, 0.935, 0.947 respectively, the sensitivity is 80.6%, 97.2%, 86.1%, 96.1%, and the specificity is 15.1%, 60.4%,

96.2%, 94.3%, P <0.05, all have statistical significance in judging the outcome of COVID-19 patients, of which the sensitivity of SAA on the 7d is the highest (Figure 2). Then the differences between the groups under the curve before and after 3d, 5d, and 7d were compared. The Z^2 results were 3.086, 5.616, and 5.671, respectively, and the P values were all <0.05,with statistical significance. It is suggested that the SAA is more meaningful to predict the prognosis of patients with prolonged treatment time.

4 Discussion

COVID-19 is a serious infectious disease caused by SARS-CoV-2. Similar to SARS and MERS,COVID-19 has atypical early symptoms [12-13], most of which are characterized by fever, dry cough, and fatigue. A few may be accompanied by symptoms such as nasal congestion, runny nose, sore throat, muscle soreness, and diarrhea. However, SARS-CoV-2 is highly contagious, and COVID-19 has posed a great threat to life and health worldwide. It is necessary to control the spread of the epidemic as soon as possible, accurately determine the prognosis of patients, and perform more effective treatment for patients with poor prognosis, such as early active and effective oxygen therapy programs. At the same time, early application of protection of important organ functions such as myocardium, kidney and liver may save the lives of more patients, which has important clinical value.

The baseline data in this study showed that the age of the non-survival group, early onset of dyspnea symptoms, and monitoring of blood oxygen saturation decreased. The non-survival group with hypertension and clustered onset were higher than the survival group. In the blood test, the white blood cell count, neutrophil count, procalcitonin, lactate dehydrogenase, urea nitrogen, creatinine, blood glucose, and D-dimer were higher in the non-survival group than in the survival group. Protein and antithrombin III activity values were lower than those in the survival group. This is similar to the clinical characteristics in 50 cases reported by Qian-Zhi Cheng et al [14]and in 62 patients with COVID-19 studied by Xu Shen et al [15], but different from those reported by Fei Zhou et al [16], which may be related to the sample size.

At present, there are very few reports about the factors affecting the prognosis of patients with COVID-19 at home and abroad. Zhang MQ et al [17] have reported that the level of lymphocytes in COVID-19 critically ill patients was generally low, and the proportion of lymphocytes was gradually decreasing, indicating a poor prognosis. Rong Qu et al [18] studied 30 cases of patients with COVID-19.The larger the platelet / lymphocyte ratio in peripheral blood during treatment, the more severe the cytokine storm, the longer the hospital stay, and the worse the prognosis. Wei Liu et al [19] reported 78 patients with COVID-19, and concluded that elevated C-reactive protein and decreased albumin were important factors affecting prognosis. The□New Coronary Virus Pneumonia Seventh Edition Diagnosis and Treatment Plan□[20] believes that the absolute count of peripheral blood lymphocytes decreased significantly, interleukin 6, C-reactive protein, increased lactic acid, and DIC screening indicators persistent abnormalities (such as D-dimer, fibrin degradation products), high levels of blood lactate dehydrogenase, and rapid progression of lesions on chest CT are all high-risk factors affecting prognosis. A retrospective analysis of the routine coagulation function of 183 confirmed COVID-19 patients by Ning Tang et al [21] showed that the

abnormal coagulation function results in death group, especially D-dimer and fibrin degradation products were significantly increased, indicating a poor prognosis. By statistical analysis of the SAA of 89 COVID-19 patients in our hospital, we believe that dynamic changes in SAA can predict the prognosis.

SAA belongs to the apolipoprotein family, mainly from the liver, plays an important role in inflammatory response and lipid metabolism, and is one of the main acute phase proteins of the body [6,9,10]. Normally, the body can secrete a small amount, but in the body after being stimulated by inflammation and trauma, it is activated by inflammatory factors such as interleukin 1 (IL-1), interleukin 6 (IL-6) and tumor necrosis factor α (TNF-α), and its secretion increases sharply, even exceeding 2,000 times the normal value, which is one of the most sensitive markers of body inflammation at present [7,8]. At present, SAA testing is commonly used in the fields of bacteria, viral infections, atherosclerosis, coronary heart disease, acute transplant rejection, and tumors. It has been reported in the literature [22] that the protein chip analysis of patients with severe acute respiratory syndrome (SARS) found that SAA may be one of the biomarkers for monitoring the degree of pneumonia and has certain value in predicting the prognosis. However, there are few reports on the value of SAA on the prognostic evaluation of COVID-19.

This study analyzes the dynamic changes of SAA in patients with covid-19, studies the correlation between SAA in different groups, before and after treatment, and draws *ROC* curves to focus on the prognosis of SAA at different time points in COVID-19 patients. The research results suggest that the protein in the acute stage SAA has a certain predictive value for the final clinical outcome of COVID-19, and its mechanism may be that SAA can activate inflammatory cells such as neutrophils, promote the release of inflammatory factors, and exacerbate inflammation in the body. At the same time,it can be combined with high-density lipoprotein (HDL) to form a SAA/HDL complex, which chemoattracts inflammatory cells; in addition, it may have an interference effect on the lipoxin signaling pathway, which can increase the survival time of neutrophils, aggravate the degree of inflammation and infection, leading to a worsening of the patient's condition [23].

In summary, this study is based on the clinical outcomes of COVID-19 patients after admission, affirming the value of SAA in the prognosis judgment of patients with COVID-19, and it is worth promoting in daily clinical work, but the sample size of this study is relatively small, leading to certain limitations of this observational study, and may lead to biased results. Therefore, multi-center, large-sample related research is the direction of future efforts. At the same time, multiple indicators such as hs-CRP, IL-6, LDH, D-dimer, and lactic acid can be jointly predicted. At present, the best diagnosis and treatment plan for COVID-19 is still under investigation. Early diagnosis and dynamic monitoring of prognostic indicators have certain value to improve the survival rate of COVID19.

Declarations

Funding

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Ethical approval

It is retrospective observational study，it was exempt from ethics approval by the ethics committee of Renmin Hospital of Wuhan University.

Consent to participate

It is retrospective observational study.

Consent for publication

All authors agree to the public publication of this article.

Availability of data and material

All collected data and material are true.

Competing interests

We have declared no competing interests.

Authors' contributions

We would like to thank the general practices and patients that participated in follow-up.

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Tables

Table 1 Comparison of general data of study population $(x \pm s)$

project	Survival group (n = 53)	Non-survival group (n = 36)	*t*/ value	*p-value*
Age (years)	53.58±15.760	68.81±12.932	-4.798	<0.001
Male / female (cases)	29/24	20/16	0.006	0.938
Heart rate (bpm)	85.13±12.639	86.50±18.434	-0.416	0.679
Respiratory rate (bpm)	19.62±2.297	20.56±5.174	-1.158	0.250
Systolic pressure (mmHg)	125.13±20.414	133.78±21.469	-1.920	0.058
Diastolic pressure(mmHg)	73.77±10.606	75.81±14.867	-0.753	0.454
SpO_2 (%)	95.28±2.656	91.53±6.222	3.908	<0.001
Body temperature (℃)	37.19±0.816	37.01±0.887	1.040	0.301
Clinical manifestation n (%)				
Fever	45（84.9）	28（77.8）	0.739	0.390
Cough	26（49.1）	14（38.9）	0.896	0.344
Expectoration	12（22.6）	8（22.2）	0.002	0.963
Sore throat	3（5.7）	0（0）	0.269[b]	0.206
Blocked nose	3（5.7）	1（2.8）	0.015[a]	0.902
Runny nose	3（5.7）	1（2.8）	0.015[a]	0.902
Chest tightness	15（28.3）	13（36.1）	0.606	0.436
Shortness of breath	8（15.1）	11（30.6）	3.052	0.081
Dyspnea	8（15.1）	12（33.3）	4.093	0.043
Fatigue	17（32.1）	19（52.8）	3.814	0.051
Basic disease n (%)				
hypertension	6（11.3）	18（50.0）	16.285	<0.001
Cardiovascular diseases	2（3.8）	5（13.9）	1.792[a]	0.181
diabetes	3（5.7）	6（16.7）	1.774[a]	0.183
COPD	2（3.8）	4（11.1）	0.854[a]	0.355
Cerebrovascular disease	1（1.9）	3（8.3）	0.845[a]	0.358
History of cancer	3（5.7）	2（5.6）	0.000[a]	1.000
History of taking hormones	4（7.5）	3（8.3）	0.000[a]	1.000
Connective tissue disease	1（1.9）	2（5.6）	0.118[a]	0.732
History of smoking n (%)	6（11.3）	5（13.9）	0.131	0.718
History of drinking n (%)	1（1.9）	1（2.8）	0.000[a]	1.000
Cluster onset n (%)	20（37.7）	5（13.9）	6.036	0.014

Note: [a] is the chi-square value of continuous correction; [b] is the Fisher test; COPD: chronic obstructive pulmonary disease, SPO_2: blood oxygen saturation

Table 2 Analysis and comparison of various indicators of the study population on the first day of admission

	Survival group (n = 53)	Non-survival group (n = 36)	*z*-value	*p-value*
Leukocyte count (10 ^ 9 / L)	4.59(3.57,5.935)	8.91(5.627,13.442)	-5.095	<0.001
Neutrophil count (10 ^ 9 / L)	2.58(2.115,4.035)	7.865(4.365,12.215)	-5.538	<0.001
Lymphocyte count (10 ^ 9 / L)	1.11(0.865,1.525)	0.71(0.435,1.087)	-3.553	<0.001
Hemoglobin (g / L)	129(116.5,140)	121.50(91.75,134)	-2.425	0.015
Platelet count (10 ^ 9 / L)	176(132,230)	185(131.25,264.25)	-0.435	0.664
C-reactive protein (mg / L)	24.0(12.5,49.6)	47.8(13,112.925)	-1.915	0.056
Serum amyloid A (mg / L)	200(87.52,200)	133.88(63.635,200)	-1.426	0.154
Procalcitonin (ug / L)	0.04(0.04,0.04)	0.194(0.069,2.111)	-6.419	<0.001
ALT/AST	0.79(0.595,1.00)	0.755(0.57,0.895)	-0.928	0.353
Albumin (g / L)	38.9(37.0,41.8)	34.6(30.4,37.1)	-5.020	<0.001
Lactate dehydrogenase (u / L)	242(175,309)	417(234,511)	-3.849	<0.001
Urea nitrogen (μmol / L)	4.02(3.405,5.08)	9.81(5.47,20.875)	-5.480	<0.001
Creatinine (μmol / L)	58(51,72)	87(57.75,182.25)	-3.734	<0.001
Blood glucose (mmol / L)	5.2(4.81,6.375)	6.285(5.072,8.655)	-2.934	0.003
GFR（ml/min）	103.55(96.83,113.35)	66.595(27.977,96.732)	-5.726	<0.001
Prothrombin time (sec)	11.8(11.3,12.25)	12.35(11.525,14.6)	-1.774	0.076
PT activity (%)	89.7(81.5,95)	78.15(58.125,92.995)	-2.512	0.012
INR	1.05(1,1.20)	1.09(0.99,1.27)	-0.280	0.779
APTT（sec）	29.8(26.8,34.635)	30.15(25.5,34.15)	-0.581	0.561
Fibrinogen (g / L)	4.05(3.29,5.045)	4.16(3.395,5.76)	-0.614	0.539
D-dimer (mg / L)	0.66(0.54,0.915)	1.61(0.677,4.49)	-4.035	<0.001
Antithrombin III activity (%)	87.8(80.5,90.615)	79.15(72.025,87.545)	-3.185	0.001

Note: ALT: alanine aminotransferase; AST: aspartate aminotransferase; GFR: glomerular filtration rate; INR: PT international standardized ratio; APTT: activated partial thrombin time.

Figures

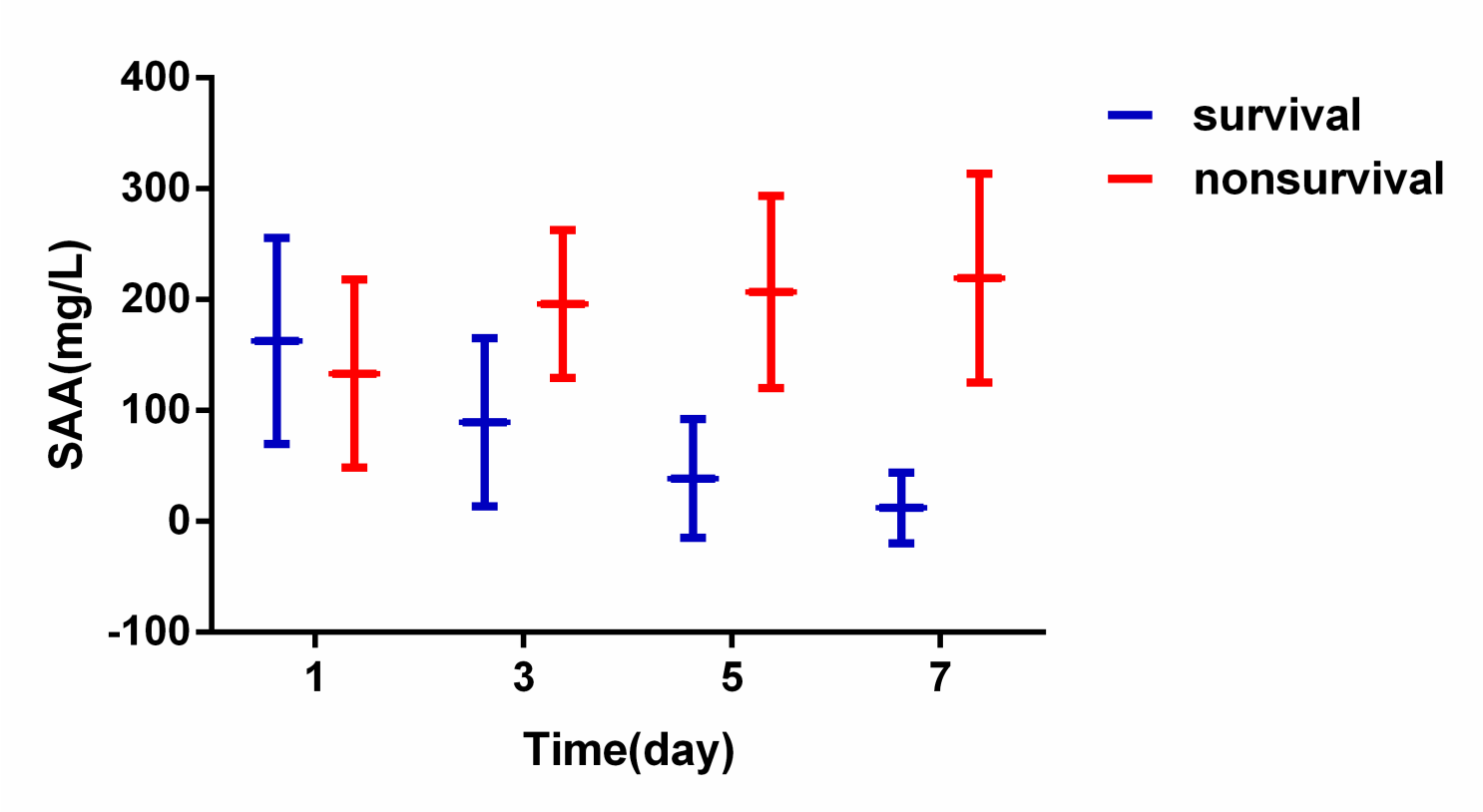

Figure 1

Comparison of serum SAA levels in survival and nonsurvival groups at various time points

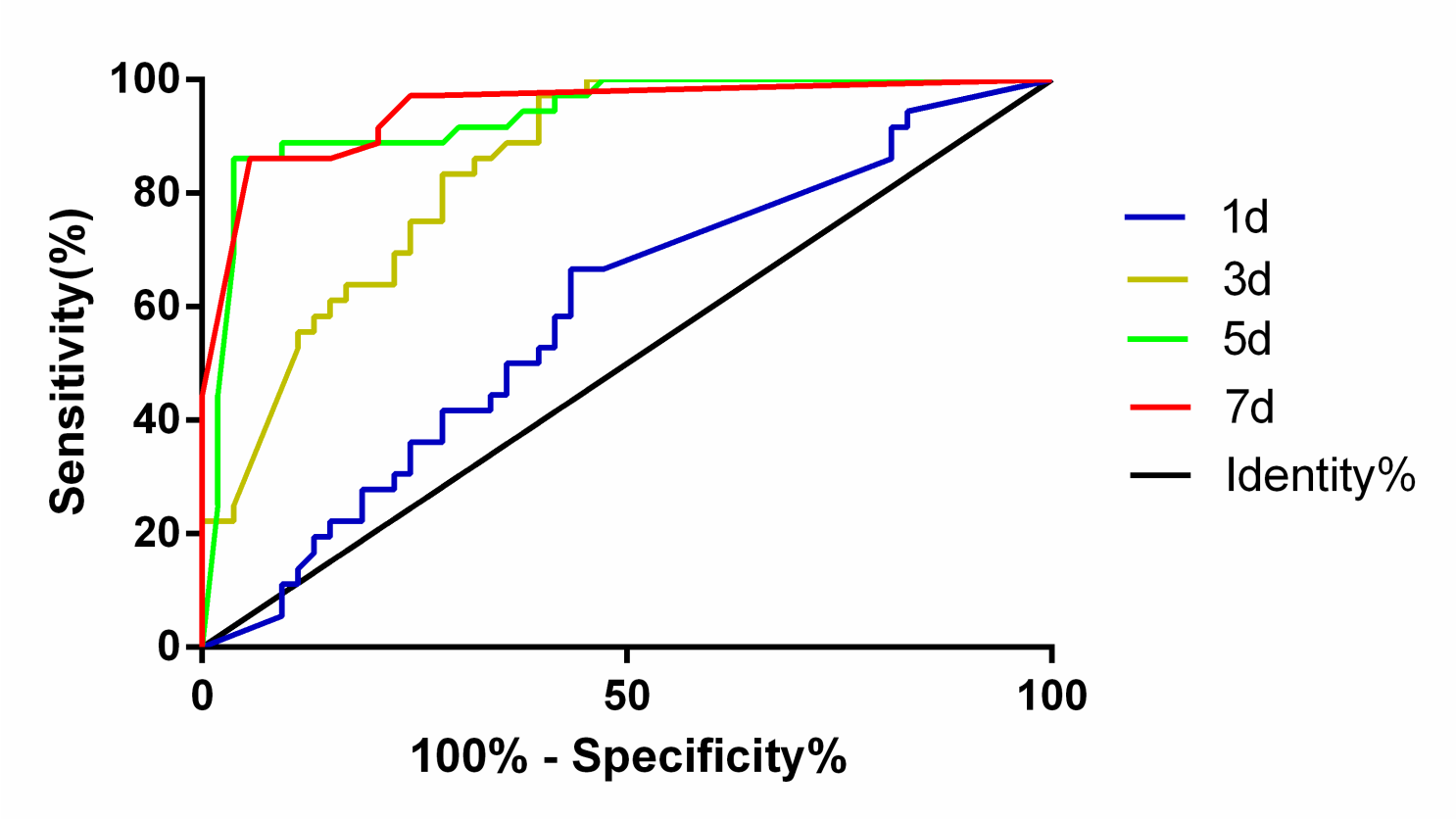

Figure 2

ROC curve of SAA against clinical outcomes of COVID-19 patients

Supplementary Files

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