Predictive factors of COVID-19 pneumonia: a retrospective cross-sectional survey in a Japanese university hospital

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Research

Keywords: COVID-19, SARS-CoV-2, pneumonia, clinical feature, computed tomography

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ABSTRACT

Background: The predictive factors of coronavirus disease 2019 (COVID-19) pneumonia, including clinical parameters with symptoms, vital signs, and laboratory data, have not been compared directly between cases with and without complicated pneumonia.

Aims: We aimed to identify predictive factors for COVID-19 patients with complicated pneumonia, and determine which COVID-19 patients should undergo computed tomography (CT).

Methods: This retrospective cross-sectional survey was conducted at the Juntendo University Nerima Hospital in Tokyo, Japan. We recruited patients diagnosed with COVID-19 between 1 January and 31 December 2020 and all patients underwent blood tests and CT. Clinical information, including vital signs, symptoms, laboratory results, and CT findings, were extracted from medical charts. Factors potentially predicting COVID-19 pneumonia were analysed using Student's *t*-test or the chi-squared test, and variables with a *p*-value of < 0.05 in the bi-variate analysis were entered into multivariate logistic regression models.

Results: Among 221 included patients (119 males [53.8%]; mean age, 54.59 ± 18.61 years), 160 (72.4%) had pneumonia. The significant factors in the multi-variate analysis were the lactate dehydrogenase (odds ratio [OR], 3.41; 95% confidence interval [CI], 1.47–7.95; $p < 0.01$) and C-reactive protein (OR, 3.94; 95% CI, 1.05–14.80; $p = 0.04$) levels. No significant differences were observed in vital signs and the symptoms.

Conclusion: LDH and CRP level of > 220 IU/L and > 3.0 mg/dL, respectively, are independent risk factors for COVID-19 pneumonia. The present results are extremely useful for deciding whether to perform CT among COVID-19 patients.

Keywords:

COVID-19; SARS-CoV-2; pneumonia; clinical feature; computed tomography

Introduction

In December 2019, a series of pneumonia cases of unknown causes were reported, involving clinical presentations that greatly resembled those of viral pneumonia [1–3]. The coronavirus disease 2019 (COVID-19) pandemic has been a great threat to human life. Many case reports on COVID-19 pneumonia have been published [4–8]. A study documented a high incidence of subclinical computed tomography (CT) changes in COVID-19 cases, with asymptomatic cases showing more ground-glass opacities (GGOs) [4]. Another study described the chest CT findings in 51 COVID-positive patients, with 77%, 75%, and 59% of cases showing pure GGOs, GGOs with interstitial and/or interlobular septal thickening, and GGOs with consolidation, respectively [8].

Furthermore, COVID-19 symptoms that don't manifest in past viral infections, such as anosmia and ageusia, have also been reported [9,10]. The wide range of reported features is thought to reflect the effects on non-respiratory systems and indicate that signs may be observed in infected patients without apparent respiratory symptoms [11].

With this, we must continuously cope with COVID-19. Given that physical examination and imaging studies, including CT, are being avoided because of the risk of nosocomial infection, predicting COVID-19 patients with complicated pneumonia as a nosocomial infection is important. Furthermore, another study suggested that due to the COVID-19 epidemic/pandemic, focusing on the exclusion of its infection using CT scans leads to an overall delay in the diagnosis and treatment of bacteremia [12]. When COVID-19 pneumonia can distinguish conclusive cases from suspicious cases, we can avoid unnecessary examinations, including CT.

However, predicting COVID-19 pneumonia is difficult, compared to other forms of pneumonia. Fever and decreased oxygenation are typical symptoms of pneumonia [13]. However, as described above, asymptomatic cases may develop critical pneumonia [4]. The presence or absence of pneumonia is clinically important, even if asymptomatic, in affecting future follow-up policies, because pneumonia is assumed to aggravate this risk. However, the

predictive factors of COVID-19 pneumonia, including clinical parameters with symptoms, vital signs, and laboratory data, have not been compared directly between cases with and without complicated pneumonia.

This cross-sectional study aimed to compare clinical parameters between patients with and without complicated pneumonia to identify predictive factors for COVID-19 patients complicating pneumonia, and determine which COVID-19 patients should undergo CT.

Methods

Study design and study population

We recruited patients diagnosed with COVID-19 from 1 January to 31 December 2020, and all patients underwent blood tests and CT. Children aged < 14 years with COVID-19 were excluded. COVID-19 was diagnosed with > 2 internists upon detection of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) nucleic acids in the polymerase chain reaction. This retrospective, cross-sectional study was performed at the Juntendo University Nerima Hospital, a 400-bed university-affiliated hospital in Tokyo, Japan.

Clinical information was extracted through chart review. Data on age, sex, history of malignant diseases, asthma, heart disease (including hypertension), diabetes mellitus, haemodyscrasia, human immunodeficiency virus infection, use of immunosuppressive agents (including steroids), and general symptoms (chills, malaise, joint pain, headache, nausea, diarrhoea, smell disturbances, taste disturbances, sore throat, cough, and difficulty in breathing) were collected. We also extracted data on axillary body temperature, blood pressure, pulse rate, respiratory rate, oxygen saturation (room air), white blood cell (WBC) count, percentages of neutrophils and lymphocytes, haemoglobin level, platelet count, red blood cell distribution width (RDW), serum parameters (total protein, albumin, lactate dehydrogenase [LDH], blood urea nitrogen, creatinine, sodium, potassium, chloride, glucose, aspartate aminotransferase [AST], alanine

aminotransferase [ALT], total bilirubin, glucose, haemoglobin A1c, and C-reactive protein [CRP] levels), and CT findings of pneumonia.

Statistical analysis

Bi-variables between COVID-19 patients with and without complicated pneumonia were compared using independent samples *t*-tests and chi-square tests for continuous and categorical data, respectively. Differences were defined as statistically significant at p values < 0.05 . Variables with a p -value of < 0.05 in the bi-variate analysis were entered into multi-variate logistic regression models, while clearly unnecessary factors in differentiation (e.g. normal electrolyte levels from the median value) were excluded. The normal or median values from bi-variate analysis determined the cut-off values. The accuracy of the logistic regression models was assessed using the area under the receiver operating characteristic curve (AUC-ROC). All statistical analyses were performed using EZR (Saitama Medical Center; Jichi Medical University, Saitama, Japan), a modified graphical user interface of R (The R Foundation for Statistical Computing, Vienna, Austria), designed to add frequently used statistical functions in biostatistics [14].

This study was conducted in accordance with relevant guidelines and regulations. This retrospective study was approved by the ethics committee of Juntendo University, Tokyo, Japan (approval number: E21-0065-N05).

Results

Among 221 included patients (119 males [53.8%]; mean age, 54.59 ± 18.61 years), 160 (72.4%) had pneumonia. Table 1 shows the patient characteristics and results from the bi-variate analysis. Age, heart disease, sore throat, difficulty in breathing, heart rate, percentages of neutrophils and lymphocytes, and albumin, LDH, AST, sodium, chloride, CRP, and glucose levels were significantly different between the two groups (Table 1). We determined age, sore

throat, difficulty in breathing, heart disease, heart rate, and neutrophil, albumin, LDH, AST, and CRP levels as clinically useful; therefore, we incorporated them in the multi-variate analysis (Table 2). Moreover, the percentages of neutrophils and lymphocytes were highly correlated with each other; thus the percentage of neutrophils was included in the multi-variate regression analysis to represent both variables. We determined each cut-off values (55 years, 85 bpm, and 3.0 mg/dL for age, heart rate, and CRP level, respectively) because of the median values of bi-variate analysis. Furthermore, the cut-offs values of 60%, 4.0 g/dL, 220 IU/L, and 30 IU/L for the percentage of neutrophils, and albumin, LDH, and AST levels were higher than the normal upper limits. The significant factors in the multi-variate analysis were the LDH (odds ratio [OR], 3.41; 95% confidence interval [CI], 1.47–7.95; $p < 0.01$) and CRP (OR, 3.94; 95% CI, 1.05–14.80; $p = 0.04$) levels. The AUC-ROC of the model was 0.792 (95% CI, 0.72–0.86), indicating sufficient power.

Discussion

We directly compared COVID-19 patients with and without pneumonia to determine which patients should undergo CT. LDH and CRP levels > 220 IU/L and > 3.0 mg/dL, respectively, were identified as independent risk factors for COVID-19 pneumonia. Furthermore, to the best of our knowledge, this is the only study that reported odds ratios (3.41 and 3.94 for LDH and CRP, respectively).

LDH is an intra-cellular enzyme found in the cells in almost all organ systems which catalyses the inter-conversion of pyruvate and lactate, with concomitant inter-conversion of NADH and NAD⁺ [15]. The enzyme is composed of subunits A and B, and is present in humans in five separate isozymes (LDH-1, LDH-2, LDH-3, LDH-4, and LDH-5 in the cardiomyocytes, reticuloendothelial system, pneumocytes, kidneys and pancreas, and liver and striated muscles, respectively). Although LDH has been traditionally used since the 1960s as a marker of cardiac damage, abnormal values can result from multiple organ injuries and decreased oxygenation

with upregulation of the glycolytic pathway. The acidic extra-cellular pH due to increased lactate from infection and tissue injury triggers the activation of metalloproteases and enhances macrophage-mediated angiogenesis [16]. Severe infections may cause cytokine-mediated tissue damage and LDH release [16]. Since LDH is present in lung tissues (LDH-3), patients with severe COVID-19 infections can release greater amounts of LDH in the circulation, often manifesting as acute respiratory distress syndrome, which is the disease hallmark [17]. Moreover, compared to non-COVID-19 pneumonia patients, COVID-19 pneumonia patients had higher AST, ALT, LDH, γ -glutamyl transpeptidase (γ -GT), and α -hydroxybutyric dehydrogenase levels [18]. Significant differences were not observed in AST, ALT, and γ -GT levels in our study; however, we similarly identified an LDH level of > 220 IU/L as an independent risk factor for COVID-19 pneumonia. Furthermore, in other studies, elevated LDH levels were associated with severe disease and mortality in COVID-19 patients [17,19, 20]. Elevated LDH levels seem to reflect that multiple organ injury and failure may play a more prominent pathological role in influencing the clinical outcomes in COVID-19 patients [17]. Therefore, the increase in LDH levels should be monitored, as it affects the overall status and disease course.

CRP is a pentameric protein synthesised by the liver, and its levels increase with inflammation. It is considered to be an acute-phase protein that responds to inflammatory cytokines associated with activated monocytes or macrophages after infection. Primarily, CRP is induced by the action of interleukin-6 responsible for CRP gene transcription. In some cases, it can also activate the complement system, forming inflammatory cytokines, thereby further aggravating tissue damage [21,22]. The CRP level correlates with CT findings and predicts severe COVID-19 early, because of significant increases in CRP levels at the initial disease stage [23]. CRP levels are also positively correlated with the diameter of the lung lesion and severe presentation [24]. We believe that these findings are consistent with our results. Furthermore, our study included cut-off values and odds ratios, which are very used in deciding whether CT should be

performed. Furthermore, daily CRP values in hospitalized COVID-19 patients can provide early thresholds and facilitate risk stratification and prognostication [25]. Therefore, It is important to be careful about changes in CRP levels, as well as LDH levels.

LDH and CRP levels may be related to respiratory function ($\text{PaO}_2/\text{FiO}_2$), and predict respiratory failure in COVID-19 patients. LDH and CRP should be considered useful for identifying patients who require closer respiratory monitoring and more aggressive supportive therapies early to avoid poor prognoses [26]. LDH and CRP are important in predicting not only the presence but also the severity of pneumonia.

In our study, no significant differences were observed in the symptoms. However, COVID-19 pneumonia may be complicated, regardless of presenting symptoms. Therefore, our study is useful because we presented laboratory data (LDH and CRP levels).

Our study had some limitations. The included population was limited to that in a single hospital. Additionally, this was a retrospective study and not argued about the variant. Therefore, we propose to conduct a multi-centre, prospective study with a larger number of patients in the future.

In conclusion, Our results are useful for differentiating COVID-19 patients with and without pneumonia. LDH and CRP level of > 220 IU/L and > 3.0 mg/dL, respectively, are independent risk factors for COVID-19 pneumonia. The present results are extremely useful for deciding whether to perform CT among COVID-19 patients.

Declarations

Ethics approval and consent to participate

This study was approved by the Ethics Committee of Juntendo University (approval number: E21-0065-N05). This study was an observational study, and written informed consent was waived due to a public health outbreak investigation of the ethics committee.

Consent for publication

All authors agreed to publish this study.

Availability of data and materials

The analysed datasets in the current study are available from the corresponding author upon reasonable request.

Competing interests

Nothing to disclose for all authors

Code availability

Not applicable

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Authors' contributions

SF, AI, MS, and TN contributed to the conceptualisation and designing of the study. SF was involved participant and data acquisition. SF, AI, MS, and TN were involved in data analysis

and interpretation. All authors were involved in manuscript preparation.

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Figure legends

Figure 1. The area under the receiver operating characteristic curve of the model was 0.792 (95% confidence interval, 0.72–0.86).

Table 1. Patient variables and results of bi-variate analysis

	Total	COVID-19 Pneumonia (+)	COVID-19 Pneumonia(-)	Name of test	<i>p</i> value
Variables	n = 221	n = 160	n = 61		
Demographic factors					
Age, years, $\pm$ SD	54.59 $\pm$ 18.61	57.67 $\pm$ 16.97	46.52 $\pm$ 20.40	t-test	< 0.001*
Female sex, n, %	102 (46.2%)	76 (47.5%)	26 (42.6%)	χ^2	0.55
Co-morbidities					
Cancer, n, %	12 (5.4%)	10 (6.3%)	2 (3.3%)	χ^2	0.52
Haemodyscrasia, n, %	6 (2.7%)	4 (2.5%)	2 (3.3%)	χ^2	0.09
Diabetes mellitus, n, %	25(11.3%)	19 (11.9%)	6 (9.8%)	χ^2	0.85
Asthma, n, %	28 (12.7%)	17 (10.6%)	11(18.0%)	χ^2	0.21
Heart disease, n, %	57(25.8%)	48 (30.0%)	9 (14.8%)	χ^2	0.03*
Human immunodeficiency virus, n, %	0	0	0	χ^2	NA
Use of immunosuppressive agents, n, %	8 (3.6%)	5 (3.1%)	3 (4.9%)	χ^2	0.81
Smoking, n, %	45 (20.4%)	31 (19.4%)	14 (23.0%)	χ^2	0.79
Symptoms					
Headache, n, %	26 (11.8%)	19 (11.9%)	7 (11.5%)	χ^2	1.00
Cough, n, %	94 (42.5%)	74 (46.3%)	20 (32.8%)	χ^2	0.10
Sore throat, n, %	32 (14.5%)	17 (10.6%)	15 (24.6%)	χ^2	0.02*

Difficulty in breathing, n, %	47 (21.3%)	40 (25.0%)	7 (11.5%)	χ^2	0.04*
Chills, n, %	8 (3.6%)	7 (4.3%)	1 (1.6%)	χ^2	0.57
Joint pain, n, %	16 (7.2%)	14 (8.8%)	2 (3.3%)	χ^2	0.27
Nausea, n, %	3 (1.4%)	1(0.6%)	2 (3.3%)	χ^2	0.38
Diarrhoea, n, %	14 (6.3%)	11 (6.9%)	3 (4.9%)	χ^2	0.82
Malaise, n, %	71 (32.1%)	48 (30%)	23 (37.7%)	χ^2	0.35
Anosmia, n, %	39 (17.6%)	27 (16.9%)	12 (19.7%)	χ^2	0.77
Ageusia, n, %	37 (16.7%)	26 (16.3%)	11 (18.0%)	χ^2	0.91
Vital signs					
Body temperature, °C, ± SD	36.93 ± 0.77	36.99 ± 0.77	36.79 ± 0.74	t-test	0.08
Systolic blood pressure, mmHg, ± SD	127.94 ± 19.53	129.10 ± 18.43	124.83 ± 22.08	t-test	0.15
Diastolic blood pressure, mmHg, ± SD	79.41 ± 14.49	80.16 ± 14.34	77.37 ± 14.83	t-test	0.21
Heart rate, bpm, ± SD	84.99 ± 15.87	86.37 ± 14.76	81.30 ± 18.15	t-test	0.03*
Respiratory rate, n, ± SD	17.26 ± 3.29	17.39 ± 3.25	16.89 ± 3.40	t-test	0.33
Oxygen saturation, %, ± SD	95.85 ± 3.60	95.62 ± 3.67	96.48 ± 3.36	t-test	0.11
Laboratory data					
White blood cell count, /μL, ± SD	5221.72 ± 2252.41	5227.50 ± 1949.74	5075.41 ± 2915.17	t-test	0.55
Neutrophils, %, ± SD	63.94 ± 13.89	65.89 ± 13.32	58.93 ± 14.20	t-test	< 0.001*
Lymphocytes, %, ± SD	26.65 ± 11.81	25.04 ± 11.06	30.78 ± 12.76	t-test	< 0.01*
Haemoglobin, g/dL, ± SD	13.98 ± 1.94	13.93 ± 1.89	14.11 ± 2.06	t-test	0.53
Red Blood cell distribution Width, %, ± SD	12.80 ± 1.44	12.76 ± 1.35	12.93 ± 1.65	t-test	0.43

Platelet, 10 ⁴ /μL, ± SD	21.66 ± 9.07	21.58 ± 9.61	21.87 ± 7.53	t-test	0.83
Blood urea nitrogen, mg/dL, ± SD	15.19 ± 10.62	15.21 ± 10.13	15.15 ± 11.90	t-test	0.97
Creatinine, mg/dL, ± SD	0.91 ± 0.68	0.88 ± 0.52	0.97 ± 0.98	t-test	0.38
Total protein, g/dL, ± SD	7.08 ± 0.68	7.05 ± 0.66	7.16 ± 0.76	t-test	0.32
Albumin, g/dL, ± SD	3.98 ± 0.61	3.88 ± 0.57	4.23 ± 0.63	t-test	< 0.001*
Total bilirubin, g/dL, ± SD	0.57 ± 0.53	0.56 ± 0.28	0.59 ± 0.90	t-test	0.70
Lactate dehydrogenase, IU/L, ± SD	244.80 ± 101.02	263.38 ± 98.13	196.36 ± 92.69	t-test	< 0.001*
Aspartate aminotransferase, IU/L, ± SD	32.33 ± 22.80	34.60 ± 24.54	26.38 ± 16.13	t-test	0.02*
Alanine aminotransferase, IU/L, ± SD	32.94 ± 38.61	35.18 ± 43.59	27.05 ± 19.51	t-test	0.16
Sodium, mEq/L, ± SD	138.45 ± 3.50	138.02 ± 3.82	139.57 ± 2.12	t-test	< 0.01*
Potassium, mEq/L, ± SD	4.07 ± 0.41	4.07 ± 0.45	4.08 ± 0.30	t-test	0.83
Chloride, mEq/L, ± SD	103.43 ± 3.84	102.96 ± 4.11	104.66 ± 2.67	t-test	< 0.01*
Creatine phosphokinase, U/L, ± SD	122.12 ± 200.44	135.85 ± 232.51	86.22 ± 48.36	t-test	0.10
Glucose, mg/dL, ± SD	114.12 ± 36.96	117.71 ± 38.78	104.53 ± 29.81	t-test	0.02*
Haemoglobin A1c, %, ± SD	6.04 ± 0.88	6.10 ± 0.90	5.90 ± 0.81	t-test	0.17
C-reactive protein, mg/dL, ± SD	3.12 ± 4.67	3.86 ± 4.67	1.18 ± 4.11	t-test	< 0.001*

**p* < 0.05

COVID-19, coronavirus disease 2019; SD, standard deviation; χ^2 , chi-square test.

Table 2. Multivariate logistic regression model

Variables	OR	95% CI	<i>p</i> -value
Age > 55 years	1.23	0.54–2.63	0.98
Sore throat	1.23	0.54–2.78	0.63
Difficulty in breathing	1.69	0.63–4.50	0.29
Heart disease	0.93	0.34–2.50	0.88
Heart rate > 85 bpm	1.06	0.51–2.20	0.88
Neutrophils > 60%	1.73	0.83–3.61	0.14
Albumin > 4.0 g/dL	0.76	0.33–1.76	0.52
Lactate dehydrogenase > 220 IU/L	3.41	1.47–7.95	< 0.01*
Aspartate aminotransferase > 30 IU/L	1.33	0.54–3.25	0.54
C-reactive protein > 3.0 mg/dL	3.94	1.05–14.80	0.04*

**p*<0.05

OR, odds ratio; CI, confidence interval

Figure 1