Original Article

Opportunity for severe and critical COVID-19 pneumonia treatment with corticosteroids: a retrospective cohort study

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Background: The coronavirus disease 2019 (COVID-19) pandemic has been the most significant infectious disease outbreak worldwide in the past 3 years, with the potential to progress to severe pneumonia and trigger systemic inflammatory response, posing a threat to human health and life. This study aims to explore the use of corticosteroids for COVID-19 and provide recommendations on the timing and dosage of the treatment.

Methods: We conducted a retrospective cohort study, enrolling 100 with COVID-19 pneumonia between December 2022 and January 2023. The diagnosis of severe and critical COVID-19 pneumonia patients was according to China's Ninth Edition of the Diagnosis and Treatment Plan for COVID-19 Pneumonia. T test and univariate proportional hazard analysis were employed to investigate the opportunity of corticosteroids therapy in relation to patients' prognosis.

Results: Compared to COVID-19 pneumonia patients treated with corticosteroids in the early phase, those who received late-phase corticosteroid therapy had a higher proportion of intensive care unit (ICU) admission (P=0.01), longer hospital stay (P=0.006), lower in-hospital survival rate (P=0.03), and slower recovery (P<0.001). A significant difference was also observed in logistic univariate proportional hazard analysis.

Conclusions: The early administration of corticosteroid therapy has been shown to significantly improve the prognosis of COVID-19 pneumonia patients, promoting recovery with significant clinical significance. Our recommendation for the administration of corticosteroid therapy is to be applied on the 6th–9th day of persisting unrelieved symptoms of COVID-19 pneumonia.

Keywords: Coronavirus disease 2019 pneumonia (COVID-19 pneumonia); corticosteroids; prognosis; hospital length of stay; survival

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Introduction

Coronavirus disease 2019 (COVID-19) pneumonia is a pulmonary inflammation condition caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infection, which has been threatening human health worldwide for the past three years, severely impacting social and economic development and limiting international communication. The diagnosis of COVID-19 infection depends on epidemiological history, clinical symptoms, and nucleic acid or antigen test results. The diagnosis of COVID-19 pneumonia is based on the appearance of specific new pulmonary imaging abnormalities in patients with COVID-19 infection (1-4). According to China's Ninth Edition of the Diagnosis and Treatment Plan for COVID-19 Pneumonia, patients are classified in accordance with the severity of the infection into four categories: mild, common, severe, and critical. Severe and critical patients require hospitalization treatment, which may include oxygen therapy, antiviral therapy, corticosteroid therapy, anticoagulant therapy, mechanical ventilation, and others.

Corticosteroids have always played a vital role in the treatment of pulmonary diseases (5). Previous researches have demonstrated that corticosteroids play a crucial role in the treatment of acute respiratory distress syndrome (ARDS) caused by viral pneumonia such as severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS) (6-9). Similarly, corticosteroids also have a significant effect on the treatment of COVID-19 pneumonia. It could significantly inhibit immune responses of pulmonary inflammatory, relieve symptoms of multi-organ failure, and increase survival rate, making it one of the important treatment options for COVID-19 pneumonia (10-15).

However, there is currently no consensus on the specific use of corticosteroids in the treatment of COVID-19 pneumonia, particularly regarding the timing and dosage of administration. This study aims to provide recommendations on the optimal timing and dosage of corticosteroid therapy based on the characteristics of COVID-19 pneumonia and clinical experience. We present this article in accordance with the STROBE reporting checklist (available at https://jtd.amegroups.com/article/view/10.21037/jtd-24-329/rc).

Methods

Study population

The single-center retrospective observational cohort study was conducted from December 2022 to January 2023 in Peking Union Medical College Hospital. Patients diagnosed as severe and critical COVID-19 pneumonia and treated with corticosteroids were enrolled. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Medical Research Ethics Committee of Peking Union Medical College Hospital (No. K23C1460) and individual consent for this retrospective analysis was waived. The inclusion criteria were: (I) at the age between 18 and 75 years; (II) treated with corticosteroids, including dexamethasone, prednisone, methylprednisolone, and hydrocortisone, etc.; (III) diagnosed as severe and critical COVID-19 pneumonia according to China's Ninth Edition of the Diagnosis and Treatment Plan for COVID-19 Pneumonia (16). The details were as follows:

- Patients who met any of the following criteria could be diagnosed as severe COVID-19 pneumonia cases:
  - SpO_2 ≤93% at rest without oxygen supplementation;
  - Respiratory distress with a respiratory rate ≥30 breaths per minute;
  - Arterial partial pressure of oxygen (PaO_2)/fraction of inspired oxygen (FiO_2) ratio ≤300 mmHg;
  - Progression of clinical symptoms with a significant increase in pulmonary infiltrates by >50% within 24–48 hours.
- Patients who met any of the following criteria could be diagnosed as critical COVID-19 pneumonia cases:
  - Respiratory failure and requiring mechanical ventilation;
  - Shock;
  - Multi-organ dysfunction syndrome necessitating ICU monitoring and treatments.

The exclusion criteria were: (I) refusal to provide clinical data, refusal to sign informed consent, or incomplete clinical data; (II) pre-existing infections from sources other than COVID-19 before infection; (III) presence of underlying immunological disorders requiring steroid treatment, such as immunosuppression, autoimmune diseases, inflammatory bowel disease, etc. Ultimately, a total of 100 patients were enrolled in this study.

Data collection

The electronic medical records of patients were

retrospectively reviewed, and demographic information, present history, past history, vital signs, auxiliary examinations, laboratory tests, corticosteroid treatments, and clinical outcome information were obtained at enrollment. After enrollment, clinical data, including vital signs and laboratory results, were recorded daily, and oxygenation was evaluated using arterial blood gases and SpO_2 measurements. The reliability of imaging examination results was independently confirmed by two radiologists who were not aware of the present study. Treatment information, including corticosteroid doses and duration, antiviral drugs, anticoagulants, antibiotics, and noninvasive respiratory support, was also documented. To assess the severity of COVID-19 pneumonia, this study used the Acute Physiology and Chronic Health Evaluation (APACHE) II scoring system, Sepsis-related Organ Failure Assessment (SOFA) score, and consciousness-urea nitrogen-respiration-blood pressure (CURB)-65 score for evaluation (17-19). In addition, other data, such as mechanical ventilation requirement, ICU admission, hospitalization time, recovery time, and survival, were also recorded.

Statistical analysis

All patients were stratified according to the median time from the first symptoms to the start of corticosteroids treatment (≤9 and >9 days). Statistical analyses were performed using SPSS statistics version 24.0 (IBM Corp., Armonk, NY, USA) and Prism 8 (GraphPad Software, La Jolla, CA, USA). Variables were tested to determine whether they had a normal distribution using the Shapiro-Wilk test and the Kolmogorov-Smirnov test. Continuous variables were expressed as mean ± standard deviation or (for non-normally distributed data) median (range) and were compared between groups using Student's *t*-test or the nonparametric Mann-Whitney *U* test. Categorical variables were presented as numbers and percentages, and clinically relevant differences were evaluated by the chi-squared test or Fischer's exact test, as appropriate. Logistic univariate proportional hazard analysis was used to evaluate the causal relationship between variables. A P value of <0.05 identified a statistically significant difference.

Results

Demographic characteristics

Table 1 summarizes the demographics and other baseline characteristics of the 100 enrolled patients. Patients treated with corticosteroids early displayed a significantly shorter duration from the first symptom onset to development of severe illness compared to those treated later (7 *vs.* 13 days, P<0.001), with a faster progression of disease. Furthermore, patients treated with corticosteroids early had higher levels of blood inflammatory cytokines, with hypersensitivity C-reactive protein (hsCRP) levels of 83.3 mg/L, significantly higher than the 62.3 mg/L observed in the late treatment group (P=0.046). This result suggested that the earlier treatment group experienced a more severe cytokine storm, and that is why they initiated corticosteroid treatments earlier according to clinical experience.

In addition to hsCRP, other indicators reflecting the inflammatory response in patients, such as procalcitonin, interleukin-6, and ferritin, also showed a similar trend of higher values in the early corticosteroid treatment group compared to the late treatment group. This suggested that although there were no guidelines or clear criteria on when to administer corticosteroids, clinicians would actively provide targeted treatment based on the actual progression of COVID-19 pneumonia, systemic inflammation and cytokine levels. Other clinical baseline indicators, such as age, gender, medical history, symptoms, APACHE II score, SOFA score, CURB-65 score, and more, showed no significant statistical differences between the two groups.

Comparison of clinical features between groups

Table 2 summarizes the hospitalization and clinical outcomes of the 100 enrolled patients. There were no significant statistical differences in the duration and dose of corticosteroid treatments between the early and late treatment groups (corticosteroid doses were expressed in an equivalent dose of prednisone, as shown in *Table 2*). Baseline vital signs and oxygenation levels also did not show any significant statistical differences between the two groups, and treatment strategies such as oxygen therapy, prone positioning, and the duration of mechanical ventilation treatment did not differ significantly between the two groups either. Additionally, there were no significant statistical differences in other treatment options, such as antiviral therapy, antibody therapy, intravenous immunoglobulin therapy, Baricitinib therapy, antibiotic therapy, antiplatelet therapy, and anticoagulation therapy, between the two groups.

However, despite the faster disease progression and more severe inflammatory response observed in patients in

Table 1 Baseline characteristics of the study population (9 days)

Characteristics	Total population (n=100)	Early corticosteroid (n=51)	Late corticosteroid (n=49)	P value
Age, years	71.5±15.1	71.9±14.9	71.1±15.5	0.81
Female	36 (36.0)	18 (35.3)	18 (36.7)	0.88
Weight, kg	68.3±11.5	68.4±10.8	68.3±12.3	0.98
BMI, kg/m^2	24.5±3.6	24.5±3.6	24.5±3.6	0.92
Comorbidities				
Hypertension	56 (56.0)	29 (56.9)	27 (55.1)	0.86
Diabetes mellitus	46 (46.0)	25 (49.0)	21 (42.9)	0.54
Cardiovascular diseases	36 (36.0)	19 (37.3)	17 (34.7)	0.79
Chronic lung diseases	22 (22.0)	12 (23.5)	10 (20.4)	0.71
Chronic renal diseases	22 (22.0)	9 (17.6)	13 (26.5)	0.28
Liver diseases	5 (5.0)	2 (3.9)	3 (6.1)	0.61
Malignancy	17 (17.0)	11 (21.6)	6 (12.2)	0.22
Smoking history	31 (31.0)	12 (23.5)	19 (38.8)	0.10
Duration from first symptoms to severe situation, d	9.5 [0.0–30.0]	7.0 [0.0–13.0]	13.0 [8.0–30.0]	<0.001
Clinical features				
Fever	95 (95.0)	47 (92.2)	48 (98.0)	0.18
Cough	95 (95.0)	48 (94.1)	47 (95.9)	0.68
Dyspnea	71 (71.0)	37 (72.5)	34 (69.4)	0.73
Chest pain	2 (2.0)	2 (3.9)	0	0.16
Headache	1 (1.0)	0	1 (2.0)	0.31
Myalgia	11 (11.0)	6 (11.8)	5 (10.2)	0.80
Asthenia	19 (19.0)	12 (23.5)	7 (14.3)	0.24
APACHE II	9.5 [2–35]	10 [2–35]	9 [3–20]	0.07
SOFA	2 [0–9]	2 [0–7]	2 [0–9]	0.30
CURB-65	1 [0–4]	1 [0–4]	1 [0–3]	0.40
Laboratory values				
White blood cells count, ×10^9/L	6.9 [1.2–23.5]	6.9 [1.2–23.5]	7.0 [1.3–20.8]	0.49
Lymphocytes count, ×10^9/L	0.7 [0.1–8.2]	0.7 [0.1–8.2]	0.8 [0.2–2.1]	0.39
HGB, g/L	118.7±24.5	119.8±25.8	117.6±23.3	0.66
Procalcitonin, ng/mL	0.2 [0.0–16.0]	0.2 [0.0–16.0]	0.2 [0.0–6.0]	0.29
ESR, mm/h	67 [18–281]	58 [22–106]	70 [18–281]	0.42
hsCRP, mg/L	70.2 [2.0–304.0]	83.3 [3.8–304.0]	62.3 [2.0–257.7]	0.046
Interleukin-6, pg/mL	13.7 [2.0–118.0]	23.0 [3.0–60.0]	6.1 [2.0–118.0]	0.77
D-dimer, mg/L	1.2 [0.2–140.1]	1.0 [0.2–72.5]	1.3 [0.2–140.1]	0.82

Table 1 *(continued)*

Table 1 *(continued)*

Characteristics	Total population (n=100)	Early corticosteroid (n=51)	Late corticosteroid (n=49)	P value
Fibrinogen, mg/dL	4.4 [1.3–9.0]	4.4 [1.3–8.2]	4.5 [1.9–9.0]	0.37
Lactate dehydrogenase, U/L	275 [157–2,173]	294.5 [193–1,024]	245 [157–2,173]	0.97
Ferritin, ng/mL	569 [126–2,183]	592 [592–592]	558.5 [126–2,183]	0.85
Creatine kinase, U/L	83 [1–3,567]	126 [1–3,567]	69.5 [1–1,178]	0.08
Troponin I, ng/mL	11.0 [2.0–8,175.0]	12.0 [3.0–8,175.0]	10.0 [2.0–2,836]	0.22
NT-proBNP, pg/mL	615 [35–35,000]	754 [35–35,000]	571 [39–35,000]	0.86
Serum creatine, μmol/L	76.5 [35.0–2,439.0]	79.0 [35.0–2,439.0]	73.0 [40.0–1,325]	0.65

Data are presented as mean ± standard deviation, n (%) or median [range]. BMI, body mass index; HGB, hemoglobin; ESR, erythrocyte sedimentation rate; hsCRP, hypersensitive C-reactive protein; NT-proBNP, N-terminal brain natriuretic peptide; APACHE, Acute Physiology and Chronic Health Evaluation; SOFA, Sepsis-related Organ Failure Assessment; CURB, consciousness-urea nitrogen-respiration-blood pressure.

the early corticosteroid treatment group (*Table 1*), and the application of the same treatment strategies in both early and late treatment groups (*Table 2*), the number of patients transferred to the ICU for treatment was significantly less in the early treatment group than in the late treatment group (2 *vs.* 10 cases, P=0.01), with a shorter duration of ICU treatment, thus showing the significant importance of early corticosteroid treatment for COVID-19 pneumonia. Moreover, the total hospitalization time was significantly shorter in the early treatment group than in the late treatment group (9 *vs.* 12.5 days, P=0.006), with a higher in-hospital survival rate observed in the early treatment group (7 *vs.* 16 cases, P=0.03), and an early recovery time (17 *vs.* 22 days, P<0.001). A significant difference was observed between the survival curves of the two groups (*Figure 1*, P=0.04), indicating that early corticosteroid therapy could effectively improve patient prognosis, shorten hospitalization time, and improve survival rates. In addition, there were no significant statistical differences in the incidence of complications related to corticosteroid therapy between the two patient groups, demonstrating the safety of corticosteroid therapy for COVID-19 pneumonia patients in a short period. Therefore, even for patients with faster disease progression and more severe systemic inflammation, early corticosteroid treatment could achieve better treatment results and prognosis.

Univariate analysis

We further conducted logistics univariate proportional hazard analysis on the clinical outcome indicators with significant statistical differences as shown in *Table 2*, to determine their causal relationship with the opportunity of corticosteroid treatment for COVID-19 pneumonia (*Table 3*). The statistical analysis revealed that there were significant statistical differences in the likelihood of being transferred to the ICU for treatment, in-hospital survival rate, and the duration time to recovery (P=0.02, 0.03, 0.005, respectively), further confirming the clinical advantages and benefits of early corticosteroid treatment for COVID-19 pneumonia.

Discussion

COVID-19 pneumonia is a respiratory disease caused by SARS-CoV-2 virus infection. The virus is a single-stranded positive-sense RNA virus with a highly variable genome of approximately 29.9 kb in length. The pathogen mainly spreads through droplet transmission, contact transmission, and aerosol transmission, and with features of a high infection rate and easy transmission. In the last 3 years, the COVID-19 pneumonia epidemic has erupted on a large scale, becoming an important global public health event (20,21).

The clinical manifestations of COVID-19 pneumonia mainly include fever, cough, fatigue, and muscle pain. Severe cases, even fatal cases, often present as severe pneumonia, ARDS, and multiple organ failure. Studies have shown that COVID-19 virus infects alveolar epithelial cells through angiotensin-converting enzyme 2 receptors, triggering cell apoptosis and inflammatory reactions, ultimately leading to lung injury and respiratory failure (20,21). Imaging examinations for the diagnosis of COVID-19 pneumonia

Table 2 In-hospital treatments and outcomes of the study population

Characteristics	Total population (n=100)	Early corticosteroid (n=51)	Late corticosteroid (n=49)	P value
Days on corticosteroid, d	6.9±2.8	6.8±2.9	7.0±2.8	0.63
Dose of prednisone (or equivalent dose of dexamethasone), mg	45.3±8.8	44.2±5.9	46.5±11.0	0.21
Vital signs				
Body temperature, ℃	38.6 [36.0–41.0]	38.5 [36.0–40.0]	38.6 [36.0–41.0]	0.34
Mean arterial pressure, mmHg	90.5 [59–130]	92 [59–130]	90 [70–121]	0.31
Heart rate, bpm	88 [57–157]	90 [60–157]	85 [57–150]	0.35
Baseline oxygenation condition				
Respiratory rate, acts/min	20 [12–40]	20 [12–40]	19 [16–35]	0.28
O_2 saturation, %	89 [39–99]	87 [50–99]	90 [39–96]	0.30
pH	7.43 [7.11–7.54]	7.43 [7.11–7.51]	7.43 [7.30–7.54]	0.40
$PaCO_2$, mmHg	33 [17–85]	33 [17–85]	34 [22–42]	0.71
PaO_2/FiO_2, mmHg	282 [107–942]	271 [146–790]	304 [107–942]	0.70
HCO_3^-, mmol/L	22.4 [14.6–38.4]	22.1 [14.6–38.4]	22.4 [15.6–27.6]	0.85
Oxygen therapy				
Low-flow oxygen	83 (83.0)	40 (78.4)	43 (87.8)	0.22
HFNC/CPAP/NIV	28 (28.0)	17 (33.3)	11 (22.4)	0.23
Mechanical ventilation	13 (13.0)	9 (17.6)	4 (8.2)	0.16
Prone ventilation	25 (25.0)	14 (27.5)	11 (22.4)	0.56
Other treatments				
Antiviral drugs	32 (32.0)	20 (39.2)	12 (24.5)	0.12
Tocilizumab	26 (26.0)	13 (25.5)	13 (26.5)	0.91
IVIG	46 (46.0)	22 (43.1)	24 (49.0)	0.56
Baricitinib	8 (8.0)	4 (7.8)	4 (8.2)	0.95
Antibiotics	77 (77.0)	43 (84.3)	34 (69.4)	0.08
Antiplatelet therapy	10 (10.0)	6 (11.8)	4 (8.2)	0.55
Anticoagulants	61 (61.0)	29 (56.9)	32 (65.3)	0.39
Length of mechanical ventilation, h	240 [17–439]	240 [17–439]	193 [55–312]	0.55
Admission to ICU	12 (12.0)	2 (3.9)	10 (20.4)	0.01
Lengthen of ICU stay, d	10 [6–22]	6.5 [6–7]	12.5 [7–22]	0.11
Hospital length of stay, d	11 [3–25]	9 [3–22]	12.5 [6–25]	0.006
In-hospital survival	77 (77.0)	44 (86.3)	33 (67.3)	0.03
Time to recovery, d	19 [3–35]	17 [3–26]	22 [12–35]	<0.001
Complications and adverse events				
Nosocomial infection	19 (19.0)	10 (19.6)	9 (18.4)	0.87
Thromboembolic events	7 (7.0)	2 (3.9)	5 (10.2)	0.22
Cardiac injury	9 (9.0)	4 (7.8)	5 (10.2)	0.68

Data are presented as mean ± standard deviation, n (%) or median [range]. FiO_2, fraction of inspiration O_2; HFNC, high-flow nasal cannula; CPAP, continuous positive airway pressure; NIV, noninvasive ventilation; IVIG, intravenous immunoglobulin; ICU, intensive care unit; $PaCO_2$, partial pressure of carbon dioxide; PaO_2, arterial partial pressure of oxygen.

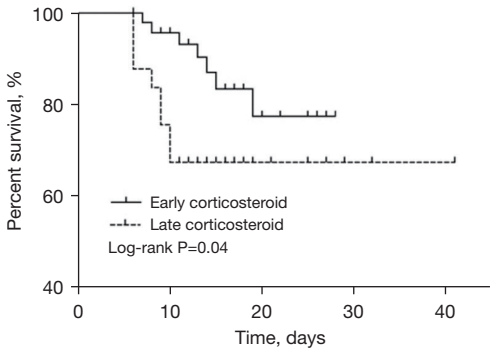

Figure 1 The survival curves of the two groups.

Table 3 Univariate proportional hazard analysis

Variable	Univariate analysis	
	HR (95% CI)	P value
Admission to ICU	6.282 (1.300–30.358)	0.02
Hospital length of stay >14 d	2.222 (0.784–6.295)	0.13
In-hospital survival	3.048 (1.125–8.254)	0.03
Time to recovery ≤21 d	0.227 (0.081–0.634)	0.005

HR, hazard ratio; CI, confidence interval; ICU, intensive care unit.

mainly rely on CT scans, which can reveal multiple, round, ground-glass nodules in bilateral lungs. Depending on the severity, COVID-19 pneumonia may also present as lung consolidation, thickening of lung markings, thickening of bronchovascular bundles, and pleural thickening (22).

The mortality rate of COVID-19 pneumonia varies greatly in different regions and age groups. The severity of the disease is also closely related to individual immune status, underlying chronic diseases, and other factors. Currently, the focus of COVID-19 pneumonia treatment is on symptomatic supportive care, aggressive rescue treatment, symptom relief, and vaccine development for prevention (20-22).

Corticosteroids, as a type of steroid medication, have long been used in the treatment of severe viral pneumonia due to their anti-inflammatory effects. In the treatment of SARS, corticosteroids are found to have certain advantages in suppressing immune response, reducing pulmonary inflammation, and decreasing mortality rates (23,24). Some early studies on the treatment of MERS also found that corticosteroids could help reduce mortality rates (25-28). However, other research has shown that the use of corticosteroids can prolong the disease course of SARS and MERS, delay the recovery from pneumonia, and increase the risk of viral infection (29-31). Additionally, corticosteroids may lead to side effects such as osteoporosis, pressure ulcers, gastrointestinal bleeding, and immunosuppression, which can affect patients' long-term recovery outcomes, and it has been shown that corticosteroids do not significantly reduce mortality rates or shorten the length of hospital stay (31). In the treatment of ARDS, some patients may benefit from corticosteroid therapy. In particular, pediatric ARDS patients are more likely to benefit from corticosteroid treatment (32,33). At the same time, the use of corticosteroids can also help reduce the inflammatory response in ARDS patients, and improve alveolar ventilation function, but it needs to be used under close monitoring (34).

The use of corticosteroids in COVID-19 pneumonia is currently one of the hot topics in research. Although corticosteroids are widely used in clinical practice, there is much controversy and uncertainty about their effectiveness, timing of use, and clinical safety. Some studies have called for caution in the use of corticosteroids and suggested that they may worsen the disease and increase mortality rates. It was reported that several severe COVID-19 pneumonia patients that the use of corticosteroids did not improve patient condition, but instead led to a trend of prolonged illness and extended virus clearance time (35-37). The National Health Institutes of the United States found in a summary analysis of COVID-19 pneumonia patient data collected from around the world that the use of corticosteroids may lead to worsening of the disease and an increase in mortality rates. However, some articles suggest that giving a certain dose of corticosteroids in the early stages of COVID-19 pneumonia treatment can help alleviate inflammation and improve patient survival rates (38,39).

Our study also suggests that giving a certain dose of corticosteroids in the early stages of COVID-19 pneumonia treatment can be beneficial. As the best hospital in China, our hospital treated a large number of COVID-19 pneumonia patients. The patient grouping in our study was formed because admitting physicians chose to use corticosteroid treatment earlier for patients with rapidly progressing and intense inflammatory responses. Although this timing of treatment mainly relied on clinical experience, the research has shown that these patients who presented with pneumonia and respiratory failure in the early stages of the disease had better outcomes with the addition of a certain dose of corticosteroid therapy. Conversely, patients who did not receive timely corticosteroid treatment were more likely

to experience respiratory failure, require ICU admission, have slow recovery, and have low survival rates, even with the use of similar doses and durations of corticosteroid treatment. Therefore, we recommend giving a certain dose of corticosteroids in the early stages of COVID-19 pneumonia treatment to alleviate the inflammatory response and achieve better prognostic outcomes.

Furthermore, our study provides specific recommendations for the opportunity and dosing of corticosteroid use. The basis of patient grouping in this study was the median timing of corticosteroid therapy: the 9th day. In the early treatment group with better treatment outcomes and prognoses, the average timing of corticosteroid use was 5.7 days after symptom onset, and the median was on the 6th day. Therefore, we recommend the use of corticosteroids on the 6–9th day after the symptom onset if it is the condition. The recommended treatment regimens are prednisone 0.60–0.75 mg/kg b.w. qd, or dexamethasone 0.09–0.11 mg/kg b.w. qd for COVID-19 pneumonia patients.

Due to the lower survival rate in the late corticosteroid treatment group in our study, treatment could not continue, thus there was no statistically significant difference in the duration of steroid treatment between the late and early treatment groups. The limitation of the present study is the small sample size. We plan to expand the sample size of this study to explore whether longer-term corticosteroid use can improve clinical prognosis in patients who use them in the late stage. Additionally, we will conduct long-term follow-up of enrolled patients to obtain data on long-term complications, survival rates, and prognosis associated with COVID-19 pneumonia and corticosteroid treatment, thus providing more clinical evidence and data support for COVID-19 pneumonia treatment.

Conclusions

The early administration of corticosteroid therapy has been shown to significantly improve the prognosis of COVID-19 pneumonia patients, promoting recovery with significant clinical significance. Our recommendation for the administration of corticosteroid therapy is to be applied on the 6-9th day of persisting unrelieved symptoms of COVID-19 pneumonia.

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Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://jtd.amegroups.com/article/view/10.21037/jtd-24-329/rc

Data Sharing Statement: Available at https://jtd.amegroups.com/article/view/10.21037/jtd-24-329/dss

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Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://jtd.amegroups.com/article/view/10.21037/jtd-24-329/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Medical Research Ethics Committee of Peking Union Medical College Hospital (No. K23C1460) and individual consent for this retrospective analysis was waived.

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