Short-Term Corticosteroid Therapy for Early Exacerbation of COVID-19 Pneumonia: A Case Report

Patient: Male, 67-year-old
Final Diagnosis: COVID-19
Symptoms: Hypoxia
Medication: —
Clinical Procedure: Short-term corticosteroid therapy
Specialty: Infectious Diseases
Objective: Management of emergency care
Background: The effect of corticosteroids in the management of patients with coronavirus disease 2019 (COVID-19) is unclear.
Case Report: A 67-year-old man who tested positive for COVID-19 by reverse-transcription PCR (RT-PCR) analysis was admitted to our hospital. On admission, he had no dyspnea and his oxygen saturation (SpO₂) level was normal. Chest imaging revealed ground-glass opacities (GGO) distributed in both lung fields. Four days after admission, bilateral lung shadows worsened, with a slight reduction in SpO₂ levels. Short-term corticosteroid therapy was initiated, and SpO₂ and radiographic findings promptly improved without use of antiviral agents.
Conclusions: More data are required to ascertain the role of corticosteroids in the management of COVID-19 pneumonia.

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MeSH Keywords: Coronavirus Infections • Pneumonia, Viral • SARS Virus • Glucocorticoids • COVID-19

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Background

Coronavirus disease 2019 (COVID-19) is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and was first reported at the end of 2019; it is now spreading worldwide [1]. On March 11, 2020, the WHO characterized COVID-19 as a pandemic [2]. A Chinese study reported that the mortality rate of COVID-19 is 2.3% in China, which is not as high as that of SARS or Middle East respiratory syndrome (MERS); however, the elderly and patients with underlying comorbidities are at a higher risk of the disease and its related mortality [3]. Although several antimicrobial agents, including arbidol [4], oseltamivir, ganciclovir [5], lopinavir/ritonavir [6,7], chloroquine [8], and remdesivir [9], have been assessed for COVID-19 treatment, no effective treatment has been established thus far. Furthermore, corticosteroid treatment is not currently recommended for COVID-19 patients [10]. Here, we present the case of a patient with COVID-19 whose condition improved with short-term systemic corticosteroid treatment early after exacerbation of pneumonia.

Case Report

A 67-year-old man who had traveled on a cruise ship was hospitalized because of fever and headache 6 days before admission. Reverse-transcription PCR (RT-PCR)-based screening for SARS-CoV-2 from a throat swab performed on the ship revealed a positive result. In accordance with national standards at the time, hospitalization was required for all positive patients. Therefore, the patient was hospitalized and monitored, without oxygen therapy. His symptoms included only fever and headache. The patient had no remarkable medical history or comorbidities except for cholecystitis. He was not receiving any long-term medication.

On admission, the patient’s vital signs included a body temperature of 37.4°C; blood pressure, 134/77 mmHg; pulse, 78/min; respiratory rate, 16/min; and oxygen saturation (SpO₂), 96% on ambient air. Physical examinations revealed no abnormalities. Laboratory investigations revealed elevated C-reactive protein (CRP, 2.89 mg/L) and white blood cell count of 3.8×10⁹/L (neutrophils, 52.5%; lymphocytes, 27.2%), with a reduction in SpO₂, to 92% on ambient air. Short-term corticosteroid therapy (i.v. administration of 125 mg methylprednisolone every 12 hours) was administered on hospitalization days 5, 6, and 7; consequently, SpO₂, and radiographic findings improved from hospitalization day 6 (Figure 1). The symptoms did not exacerbate after discontinuation of corticosteroid treatment. However, despite improvement in SpO₂, the patient could not be discharged owing to the positive result of an RT-PCR test performed on hospitalization day 14. Finally, RT-PCR tests of nasopharyngeal swabs and throat swabs yielded negative results on hospitalization days 16 and 17. The patient was then discharged on hospitalization day 18.

Discussion

This study reports the case of a COVID-19 patient with improved overall conditions after short-term systemic corticosteroid treatment early after the exacerbation of pneumonia. Our findings suggest that systemic corticosteroid therapy may be a potential treatment alternative for exacerbated COVID-19 pneumonia. Moreover, the present case potentially provides novel insights into the application of corticosteroids for treating COVID-19.

In some respiratory infections, a mortality benefit has been achieved through timely administration of corticosteroids [11,12]. A meta-analysis by Siemieniuk et al. reported that systemic corticosteroid therapy can reduce mortality in community-acquired pneumonia requiring hospitalization [13]. Moreover, corticosteroids co-administered with anti-Pneumocystis cystis therapy can decrease the mortality rate and respiratory failure associated with Pneumocystis pneumonia among patients with human immunodeficiency virus infections [12]. However, the benefits of corticosteroid treatment in influenza remain unknown [14]. Corticosteroids were often prescribed empirically to SARS and MERS patients [15,16]. However, corticosteroid administration for COVID-19 patients is not recommended thus far; this includes patients with severe pulmonary injury or shock, based on evidence from other viral infectious diseases, including SARS or MERS [10]. In the present case, SpO₂, and radiographic findings improved the day after systemic corticosteroid treatment was initiated, with no exacerbation of respiratory status or pneumonia, after discontinuation of corticosteroid treatment. Administration of lopinavir/ritonavir was considered if pneumonia had not improved the day after the initiation of corticosteroid therapy. However, the patient’s condition improved without additional treatment, suggesting that some COVID-19 patients can benefit from systemic corticosteroid therapy.

However, it remains unclear which COVID-19 patients benefit from corticosteroid treatment. Sung et al. reported that

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Table 1. Laboratory data on admission.

| Complete blood count         | Blood chemistry                  |
|-----------------------------|----------------------------------|
| White blood cell count       | Aspartate aminotransferase       |
| Neutrophil                  | 3800 /μg                        |
| Eosinophil                  | 51.4 %                           |
| Lymphocyte                  | 0.4 %                            |
| Red blood cell count         | 16.5×10⁴ /μl                     |
| Hemoglobin                  | 471×10⁴ /μl                      |
| Hematocrit                  | 13.9 g/dl                        |
| Platelet count              | 42.8 %                           |
| Total protein               | 16.5×10⁴ /μl                     |
| Hemoglobin                  | 6.4 g/dl                         |
| Hematocrit                  | 3.9 g/dl                         |
| Platelet count              | 242.8 %                          |
| Total protein               | 3.9 g/dl                         |
| Hemoglobin                  | 13.6 mg/dl                       |
| Hematocrit                  | 0.7 mg/dl                        |
| Platelet count              | 102 mg/dl                        |
| Total protein               | 6.1 %                            |

APTT – activated partial thromboplastin; PT – prothrombin time.

Figure 1. Clinical course of the patient. Chest X-rays revealed worsening diffuse infiltrates (arrow) before systemic corticosteroid therapy. After administration of systemic corticosteroid therapy on days 5–7, chest X-ray images and SpO₂ levels displayed immediate and sustained improvement. SpO₂ – oxygen saturation; BT – body temperature.
the administration of corticosteroids to patients with SARS, relatively early after onset, yielded a favorable outcome [15]. Previous studies have reported that in acute viral respiratory infections, early-response cytokines, including interferon-tumor necrosis α, interleukin (IL)-1, and IL-6, are produced at high levels and mediate antiviral activity; however, they simultaneously potentially contribute to tissue injury [17,18]. Early corticosteroid therapy among SARS patients may have prevented death by regulating cytokine responses [15]. Similarly, in the present case, early corticosteroid treatment may have suppressed such an inflammatory burst in a COVID-19 patient, thus preventing the need for ventilator management. However, corticosteroid treatment was reportedly ineffective among MERS patients in an intensive care unit; the general condition of the patients was not serious enough to warrant admission to the intensive care unit, whereas previously reported subjects had a severe pathological condition [16]. The present case report included only 1 patient and the patient’s condition might have improved spontaneously without corticosteroid treatment. Although the exact role of corticosteroid treatment cannot be determined owing to limited data, corticosteroid treatment in the early stage of COVID-19 pneumonia exacerbation may be considered a potential treatment alternative.

**Conclusions**

Our findings indicate that corticosteroid treatment can benefit cases of mild and early-stage exacerbation of pneumonia among COVID-19 patients; however, this was an uncontrolled observation and warrants further controlled clinical trials. Additional cases need to be studied to determine the precise symptoms of COVID-19 patients who would benefit most from systemic corticosteroid therapy.

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**Conflict of interest**

None.

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**Figure 2.** Chest computed tomography images on admission revealed bilateral ground-glass opacity (GGO) in subpleural areas (arrow).
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