Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Respiratory Investigation

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Editorial

SARS-CoV-2 as a causative agent of idiopathic interstitial pneumonia and interstitial pneumonia associated with collagen vascular disorders

Keywords:
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Autoimmune interstitial pneumonia

Fluid accumulation in the lungs due to coronavirus disease (COVID-19)-associated pneumonia is a result of inflammation caused by the secretion of inflammatory chemokines such as tumor necrosis factor (TNF-α), released by cells of the immune system. This can eventually lead to acute respiratory distress syndrome (ARDS), which is the primary cause of mortality in COVID-19 patients. Examination of pathological specimens collected from patients with ARDS frequently reveals diffuse alveolar damage. Moreover, laboratory studies have demonstrated both alveolar epithelial and lung endothelial injury, resulting in accumulation of protein-rich inflammatory edematous fluid in the alveolar space [1].

Murohashi et al. presented 11 cases wherein severe COVID-19-related pneumonia was treated with favipiravir and methylprednisolone [2]. They concluded that early-stage treatment using a combination of favipiravir and methylprednisolone could prevent the need for ventilatory support [2]. Although there is currently no definite treatment for COVID-19, clinical trials are underway to determine potential treatments using existing drugs. Researchers conducting the Randomised Evaluation of COVID-19 Therapy (RECOVERY) Trial, currently underway in the UK, announced that dexamethasone had shown considerable potential in significantly improving the outcomes for COVID-19 patients receiving respiratory support [3].

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I was further impressed with the findings reported in the article by Murohashi et al. [2], particularly regarding the radiological features of case 10. Before December 2019, we were not aware of the clinical features and radiological findings of COVID-19 patients. Without this knowledge, I would have diagnosed case 10 as nonspecific interstitial pneumonia (NSIP) [4] or autoimmune interstitial pneumonia (AIIP) [5].

From a case series we conducted at the University of the Ryukyus Hospital, I present the cases of three patients whose chest computed tomography (CT) images are shown in Fig. 1. Chest CT findings of case 1 demonstrated typical pneumonia caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) on days 1 and 3 after admission; however, on day 9, they indicated organizing pneumonia (OP). Similarly, chest CT findings of case 2 demonstrated typical pneumonia caused by SARS-CoV-2 on day 1. However, on day 7, they indicated possible NSIP or AIIP, which closely resembled findings of case 10 in the article by Murohashi et al. [2]. Chest CT findings of case 3 showed a pattern of "radiographic negative for pulmonary edema", indicative of OP or chronic eosinophilic pneumonia. Because these conditions were diagnosed as COVID-19-associated pneumonia, pathological evaluation was not performed. However, previously reported pathological findings of COVID-19-associated pneumonia have demonstrated variations from those of OP, interstitial pneumonia/fibrosis, and diffuse alveolar damage [6,7]. Additionally, several reports have described the similarity between pneumonia caused by SARS-CoV-2 and interstitial pneumonia

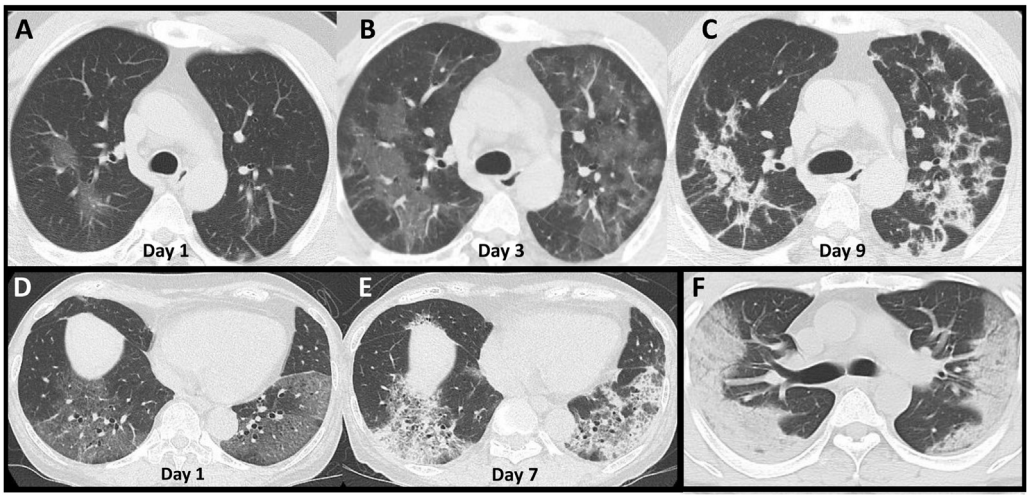

Fig. 1 – Chest computed tomography (CT) results of 3 cases of pneumonia caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). A–C: A 42-year-old man, CT images on days 1, 3, and 9 after admission. D–E: A 68-year-old woman, CT images on days 1 and 7 after admission. F: A 45-year-old man, CT image on day 1 after admission.

associated with collagen vascular diseases (IP-CVD) [8,9]. Furthermore, Megremis et al. identified three immunogenic linear epitopes with high sequence identity to SARS-CoV-2 proteins in patients with dermatomyositis [10].

Because pneumonia caused by SARS-CoV-2 is similar to idiopathic interstitial pneumonia (IIP) and IP-CVD, I would diagnose these conditions as OP, NSIP, or AIIP, and definitively choose steroid therapy for treatment. In addition, SARS-CoV-2 might provide clues to the pathogenesis of IIP or IP-CVD. Despite the uncertainty surrounding the many pathogenetic aspects of COVID-19-related pneumonia, we are probably observing a model for other forms of IIP or IP-CVD [9,10]. The large amount of data derived from studies on COVID-19 could assist in understanding the pathogenesis of IIP or IP-CVD and developing new therapeutic strategies.

Conflict of Interest

The author declares no conflict of interest.

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