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PIV PEDIATRIC INFECTION & VACCINE

Case Report



# Unexpected Aggravation of COVID-19 After Recovery in Three Adolescents With Chronic Neurologic Conditions: A Case Series

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No potential conflict of interest relevant to this article was reported.

## ABSTRACT

The clinical severity of coronavirus disease 2019 (COVID-19) in children is usually mild. Most of the affected patients completely recovered from COVID-19 before being released from approximately 7-day quarantine. However, children with comorbidities are at risk of more severe disease and adverse outcomes. We report three cases of COVID-19-affected adolescents with underlying chronic respiratory difficulty due to neurologic diseases who showed sudden clinical aggravations at the time of discharge, even after full clinical improvement. Patient 1 is a 17-year-old boy with Ullrich congenital muscular dystrophy who had cardiopulmonary arrest 9 days after the initial COVID-19 symptoms. Patient 2 is a 17-year-old girl with intracerebral hemorrhage with infarction in bed-ridden status who had cardiopulmonary arrest 11 days after the initial symptoms. Patient 3 is a 12-year-old boy with intraventricular hemorrhage with hydrocephalus in bed-ridden status who showed multiorgan failure 10 days after the initial symptoms. Remdesivir, dexamethasone, and empirical antibiotics were administered with mechanical ventilation and intensive unit care. Among the three patients, two (patients 1 and 3) were alive, and one (patient 2) expired. Clinicians caring for adolescents with chronic neurologic and/or pulmonary disease should keep in mind that these patients could have sudden deterioration after recovery from the acute phase of COVID-19 around or after the time of discharge.

**Keywords:** COVID-19; Adolescent; Respiratory insufficiency; Chronic disease

## INTRODUCTION

The coronavirus disease 2019 (COVID-19) pandemic has resulted in millions of affected patients, including children.[1,2] Most affected children have a mild clinical course and do not need hospital care.[3] Although a few showed a severe initial clinical presentation requiring intensive unit care or mechanical ventilation, only a small portion of affected children needed readmission after release from quarantine because of aggravated symptoms of COVID-19 and/or underlying disease.[4,5] Even high-risk patients are mostly discharged after successful recovery from COVID-19, and these patients are not always actively followed up in the outpatient clinic.[6] However, we report three pediatric cases of COVID-19, in which the patients showed sudden clinical aggravations around the time of discharge, even after full clinical improvement while quarantined.

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**Author Contributions**

Conceptualization: Yun KW. Data curation: Kang DY, Song SH, Ahn B, Lee BJ. Formal analysis: Kang DY, Song SH, Ahn B. Investigation: Kang DY. Writing - original draft: Kang DY, Yun KW. Writing - review & editing: Song SH, Ahn B, Lee BJ.

## CASE

### 1. Patient 1

On November 24, 2021, a 17-year-old boy complained of fever, cough, sputum production and dyspnea, and he needed the use of home mechanical ventilation via a nasal mask without $O_2$ supply all day, which he used to use only at night. He was diagnosed with COVID-19 via SARS-CoV-2 real-time reverse-transcriptase polymerase chain reaction (RT-PCR) and was admitted to our hospital the next day. He had been previously diagnosed with Ullrich congenital muscular dystrophy, had been wheelchair bound and had been using a bilevel positive airway pressure (BiPAP) via nasal mask for 8 years.

The initial examination showed vital signs that included normal blood pressure (BP) with a high heart rate (HR) of 144 beats per minute (bpm) and mild fever of 37.8°C. The laboratory results showed a white blood cell (WBC) count of 22,730/μL (lymphocytes 6%) and C-reactive protein (CRP) of 10.76 mg/dL. Chest computed tomography (CT) suggested bronchopneumonia (**Fig. 1A**). Ampicillin/sulbactam and dexamethasone were started. On day 8, the fever subsided, and the laboratory findings greatly improved, with a WBC count of 6,450/μL (lymphocytes 14.4%) and CRP of 5.39 mg/dL. However, he still had a large amount of secretion requiring frequent suction.

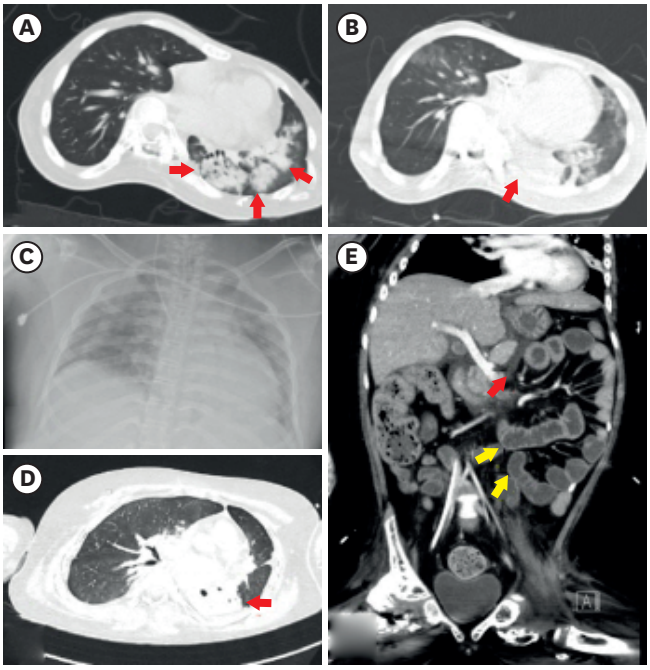


**Fig. 1.** Radiographic findings in three adolescents with coronavirus disease 2019. (A) Chest CT findings of patient 1 one day after the initial symptom; peribronchial consolidation in LUL lingular division and LLL field suggesting r/o bronchopneumonia (red arrows). (B) Chest CT findings of patient 1 four days after the CPR event; patchy ground glass opacities in right upper lobe, right middle lobe, LUL field with atelectasis in LLL field (red arrow). (C) Chest X-ray findings of patient 2 on the day of CPR event; patchy consolidation in bilateral upper lobe and right lower lobe suggesting r/o pneumonia. (D) Chest CT findings of patient 3 on the day of symptom aggravation (10th day from the initial symptom); atelectasis in LLL (red arrow). (E) Abdomen CT findings of patient 3 on the day of symptom aggravation (10th day from the initial symptom); small amount of peripancreatic fluid collection (red arrow), diffuse fluid filled, dilatated small bowel loops with mild bowel wall thickening (yellow arrows). Abbreviations: CT, computed tomography; LUL, left upper lobe; LLL, left lower lobe; CPR, cardiopulmonary resuscitation.


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On day 9, a sudden cardiopulmonary arrest occurred unexpectedly, and cardiopulmonary resuscitation (CPR) was performed for 10 minutes. Post-CPR laboratory results showed an elevated WBC count of 16,820/μL (lymphocytes 21.9%) and aspartate aminotransferase/alanine transaminase (AST/ALT) of 445/277 IU/L. Cardiac enzymes were normal with creatine kinase MB (CK-MB) isoenzyme of 0.8, troponin I of below 0.01, and B-type natriuretic peptide (BNP) of 11. D-dimer was not evaluated. Echocardiography showed acceptable left ventricle contractility with minimal left ventricular hypertrophy (ejection fraction 64.5%). No microorganism was found in blood culture, respiratory bacterial culture and respiratory virus polymerase chain reaction (PCR). Chest CT showed multiple patch ground glass opacities with atelectasis in both lungs (**Fig. 1B**). Intubation was not performed, but a home mechanical ventilator via a nasal mask was applied with increased inspiratory positive airway pressure and sufficient $O_2$ supply. Vancomycin, piperacillin/tazobactam, and remdesivir were started. On day 13, the patient's clinical status was much stabilized, and then the daytime home mechanical ventilation was discontinued on day 22 (**Fig. 2A**).

## 2. Patient 2

On February 23, 2022, a 17-year-old girl visited our hospital due to fever, sputum and dyspnea that lasted for 3 days and was diagnosed with COVID-19. When she was 10 years old, she had a sudden right basal ganglia hemorrhage with occipitotemporal lobe infarction requiring craniotomy and frontotemporal lobectomy. After the event, tracheostomy with a home-ventilator machine and percutaneous endoscopic gastrostomy (PEG) tube were applied, and she became bed-ridden.

The laboratory tests at the initial visit showed WBC of 9,080/μL (lymphocytes 19.1%), elevated CRP levels up to 10.49 mg/dL and AST/ALT levels up to 201/287 IU/L. The initial chest X-ray showed no abnormalities. The fever subsided on day 5, and the CRP level decreased to 1.15 mg/dL with AST/ALT of 176/152 IU/L. Therefore, she was discharged on day 6.

Two days after discharge, she visited the emergency department due to oxygen desaturation. Her BP dropped to 86/60 mmHg, her HR increased to 153 bpm, her body temperature (BT) reached 41.2°C, and her saturation of percutaneous oxygen ($SpO_2$) was 80%. The laboratory results showed a WBC count of 12,550/μL (lymphocytes 29.9%), CRP of 24.48 mg/dL, procalcitonin (PCT) of 0.409 ng/mL and D-dimer of 6.99 μg/mL (FEU) (reference range 0.04-0.49 μg/mL [FEU]). Blood culture showed no microorganism, and respiratory bacterial culture showed rare amount of *Pseudomonas aeruginosa*. Chest X-ray showed pneumonic infiltrations in both lungs (**Fig. 1C**). Continuous inotropic infusion, inhaled nitric oxide with a sufficient fraction of inspired oxygen ($FiO_2$), and continuous renal replacement therapy (CRRT) were started. Although she required invasive mechanical ventilation and showed mild renal failure with an eGFR (estimated glomerular filtration rate) of 57 mL/min/1.73 $m^2$, remdesivir at the usual dose with dexamethasone and teicoplanin plus meropenem was started considering the risks and benefits. Despite aggressive medical therapies, metabolic acidosis rapidly progressed. The next day, two attacks of sudden cardiopulmonary arrest occurred. The following cardiac biomarkers were elevated: CK-MB isoenzyme up to 91.9 ng/mL, troponin I up to 31.57 ng/mL, myoglobin over 12,000 ng/mL, and BNP up to 4,472 pg/mL. Echocardiography showed severe concentric left ventricular hypertrophy with severe dysfunction and a moderate amount of pericardial effusion (ejection fraction 37.7%). Intravenous immunoglobulin (IVIG) was started. Extracorporeal membrane oxygenation treatment was considered, but her family did not want further management, and the patient expired (**Fig. 2B**).


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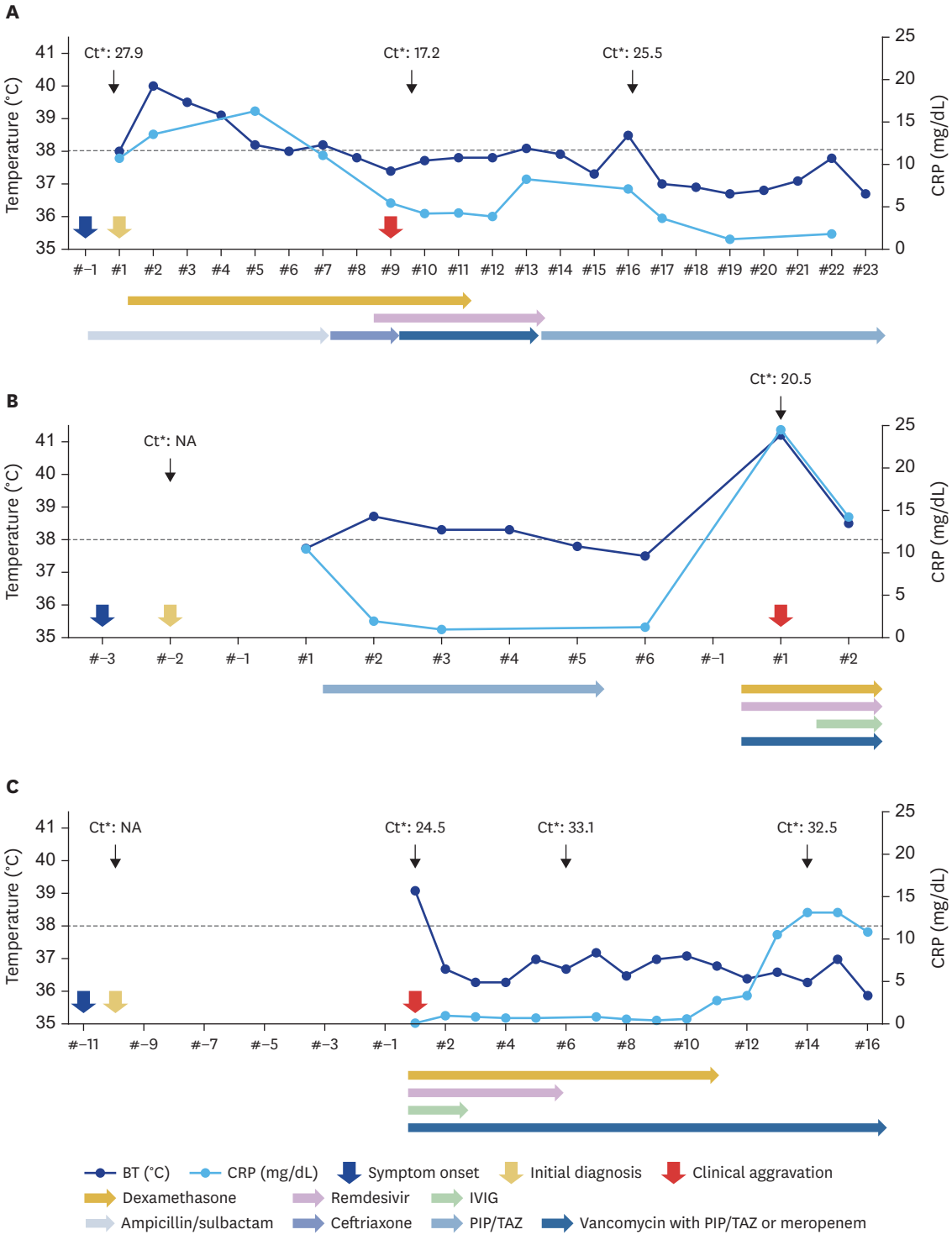


**Fig. 2.** The clinical summary of three adolescent patients with coronavirus disease 2019 who worsened to develop critical disease. (A) Clinical summary of patient 1; cardiopulmonary arrest occurred 9 days after the first symptom due to mucus obstruction followed by inadequate toileting. (B) Clinical summary of patient 2; cardiopulmonary arrest occurred 11 days after the first symptom due to acute heart failure with fulminant myocarditis. (C) Clinical summary of patient 3; multiorgan failure occurred 10 days after the first symptom.
Abbreviations: Ct, cycle threshold value in SARS-CoV-2 reverse-transcriptase polymerase chain reaction; CRP, C-reactive protein; NA, not available; BT, body temperature; IVIG, intravenous immunoglobulin; PIP/TAZ, piperacillin/tazobactam; SARS-CoV-2, severe acute respiratory syndrome-coronavirus-2.
*Ct value of RdRp gene. The specimens for all SARS-CoV-2 polymerase chain reactions were from nasopharyngeal swabs.


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### 3. Patient 3

On March 13, 2022, a 12-year-old boy complained of fever for three days with cough and sputum production and was diagnosed with COVID-19. He was not admitted to the hospital because the symptoms were mild. He was born as a preterm baby with a gestational age of 24 weeks and a birth weight of 680 g and diagnosed with intraventricular hemorrhage grade IV with hydrocephalus and periventricular leukomalacia, for which he received a ventriculoperitoneal shunt insertion. He was taking antiepileptic drugs for frequent seizures and was in a bed-ridden status with a PEG tube for feeding. He showed short stature and failure to thrive with a height of 122 cm (<1 percentile) and weight of 25 kg (<1 percentile).

On the 10th day from the initial symptom, he fell into a sudden coma even though his clinical course was improving just before the incidence. His BP dropped to 67/44 mmHg, with an HR of up to 179 bpm. His BT was 39.1°C, his respiratory rate was 46 bpm, and his $SpO_2$ was 66%. The laboratory results showed a WBC count of 22,740/μL (lymphocytes 30%), CRP of 0.17 mg/dL, and PCT 44.01 ng/mL. The pH of VBGA was 6.98, $pCO_2$ 57 mmHg, base excess -18.2, and lactic acid 9.3 mmol/L. Myocarditis was suspected with a CK-MB of 83.4 ng/mL, a troponin I of 1.84 ng/mL, and a decreased ejection fraction of 48% on echocardiography. Rhabdomyolysis was suspected with creatine kinase of 10,740 IU/L, myoglobin over 12,000 ng/mL, aldolase over 100 U/L, and AST/ALT of 6,329/4,096 IU/mL. Disseminated intravascular coagulation was observed with a D-dimer of more than 138 μg/mL (FEU), increased prothrombin time-international normalized ratio of 4.03, increased activated partial thromboplastin time of 106.4 sec, and decreased fibrinogen of 52 mg/dL. Blood and cerebrospinal fluid cultures, respiratory virus PCR showed no microorganisms. Respiratory bacterial culture showed rare amount of methicillin-resistant *Staphylococcus aureus*. Atelectasis in the left lower lung field was observed on chest CT (**Fig. 1D**). Pancreatitis and enteritis were also suspected, with increased amylase/lipase up to 1,707/902 U/L and peripancreatic fluid collection with bowel wall thickening on abdominal CT findings (**Fig. 1E**). Mechanical ventilation after intubation, CRRT, and aggressive transfusion therapy were performed. Although he showed high AST/ALT levels, remdesivir at the usual dose with dexamethasone was started considering the risks and benefits. IVIG with vancomycin plus meropenem was also started. After 16 days of hospitalization, the patient's general condition improved, and he was transferred to an outside hospital for the control of shunt malfunction (**Fig. 2C**). We compared each patient's baseline characteristics and clinical features in **Table 1**.

### 4. Ethics statement

This study was approved by the Institutional Review Board (IRB) of Seoul National University Hospital, and the requirement for informed consent was waived (IRB No. 2206-138-1335).

## DISCUSSION

In children, the clinical severity of COVID-19 is often mild, and the acute symptoms usually improve within a couple of weeks.[3,7,8] However, the patients in our study showed quite different clinical courses from the usual course. In case 1, airway obstruction by the mucus plugs due to insufficient airway toileting and inefficient lung care might have been the main cause of cardiorespiratory arrest. In case 2, acute heart failure with fulminant myocarditis was the cause of cardiorespiratory arrest. In case 3, multiple organ failures were supposed to be the main cause of clinical and laboratory aggravations. Any secondary viral, bacterial, or fungal infection did not seem to be the cause of clinical aggravation in all three patients,


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**Table 1.** Demographics, clinical features, treatments and outcomes in the three coronavirus disease 2019 patients

| Variables | Patient 1 | Patient 2 | Patient 3 |
|---|---|---|---|
| Age (yr) | 17 | 17 | 12 |
| Sex | M | F | M |
| Body weight | 30 kg | 62 kg | 25 kg |
| Underlying disease | Ullrich congenital muscular dystrophy | ICH with infarction | IVH with hydrocephalus and PVL |
| Underlying state | BiPAP via nasal mask during nighttime<br>Wheelchair bound | Bed-ridden state<br>Tracheostomy on home-ventilator<br>PEG tube insertion state | Bed-ridden state<br>PEG tube insertion state<br>Seizure on AED |
| $O_2$ requirement before aggravation | Room air | 3 L/min | Room air |
| Days after the initial symptom | 9 | 11 | 10 |
| Initial symptom | Fever, URI, dyspnea | Fever, URI, dyspnea | Fever, URI |
| Symptoms at aggravation | Cardiopulmonary arrest due to excessive sputum and atelectasis | Cardiopulmonary arrest due to acute heart failure with fulminant myocarditis | Multiorgan failure (myocarditis, rhabdomyolysis, DIC, pancreatitis, enteritis) |
| Maximum $O_2$ requirement at aggravation | 15 L/min | $FiO_2$ 80% | $FiO_2$ 40% |
| Treatment | Remdesivir, Dexamethasone, Antibiotics<br>Noninvasive mechanical ventilator | Remdesivir, Dexamethasone, Antibiotics<br>Mechanical ventilator, CRRT | Remdesivir, Dexamethasone, Antibiotics<br>Mechanical ventilator, CRRT |
| Outcome | Alive | Expired | Alive |

Abbreviations: M, male; F, female; ICH, intracerebral hemorrhage; IVH, intraventricular hemorrhage; PVL, periventricular leukomalacia; BiPAP, bilevel positive airway pressure; PEG, percutaneous endoscopic gastrostomy; AED, antiepileptic drugs; URI, upper respiratory infection; DIC, disseminated intravascular coagulation; CRRT, continuous renal replacement therapy.

based on the follow-up cultures and respiratory virus RT-PCR. Thromboembolism was not fully evaluated and excluded, despite some suspicious clinical findings were shown. The clinical aggravation of these patients was not explained by bacterial coinfection or aspiration pneumonia considering radiologic and microbiologic test results. The courses of these patients were less likely explained by MIS-C because the onset time was shorter than 2 weeks and there were no gastrointestinal or mucocutaneous symptoms that are frequently accompanied in MIS-C.[9-11] Fungal culture or galactomannan test for *Aspergillus* was not examined, but all those patients were not immunocompromised and there were no radiologic findings suggesting invasive pulmonary aspergillosis.

Two theories of the pathophysiology of COVID-19 have been described. The first mechanism is that SARS-CoV-2 penetrates the host cell via angiotensin converting enzyme-2 (ACE2) and destroys the main organs, such as the lungs, heart, ileum, and kidney, which show high ACE2 expression.[1,12] The second mechanism is that SARS-CoV-2 triggers an excessive immune response leading to a cytokine storm that can damage vital organs.[1,13,14] The fact that pediatric patients show milder symptoms than adults because of less maturation of ACE2 and a low incidence of cytokine storms supports this pathophysiology.[1,15] Therefore, the severity of COVID-19 seems to be associated with the degree of underlying immune response in each patient.[14] The three patients in our study showed sudden clinical aggravations approximately 10 days after infection with COVID-19. Even though no clear mechanism has been identified, we assumed that reactivation of SARS-CoV-2 itself or the immune response to the virus triggered hyperinflammation and caused a cytokine storm leading to multiorgan failure with cardiorespiratory arrest.

Children with certain underlying diseases can have more severe symptoms than healthy children when affected by COVID-19.[1,16] In particular, children with a neurologic or respiratory disease commonly show aggravation of symptoms associated with the underlying disease.[16] In our three cases, all patients had chronic respiratory difficulty caused by neurologic diseases that resulted in respiratory difficulties during aggravation. Currently, prophylactic remdesivir for 3-day is recommended in high-risk patients who have underlying


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disease such as chronic lung disease to prevent progression to severe COVID-19. In our study, patient 1 had been diagnosed before the approval of prophylactic remdesivir and patient 3 was not eligible for this prophylactic therapy due to his low body weight (25 kg). The only eligible patient 2 had not received prophylactic remdesivir, but the role of prophylactic remdesivir on the prevention of the clinical aggravation for these patients is not certain.

In conclusion, we experienced three critical pediatric COVID-19 cases who showed sudden clinical aggravations at the time of discharge, even after full clinical improvement. All of these patients were adolescents between 12–18 years old and had chronic respiratory difficulty due to underlying neurologic diseases. Even if it is time to release the patient from quarantine, careful monitoring of the patient is still needed to screen for clinical aggravation, particularly in those patients with underlying neurologic and/or airway disease.

## REFERENCES

1. Castellar-López J, Villamizar-Villamizar W, Amaranto-Pallares A, Rosales-Rada W, De Los Angeles Vélez Verbel M, Chang A, et al. Recent insights into COVID-19 in children and clinical recommendations. Curr Pediatr Rev 2022;18:121-37.
PUBMED | CROSSREF
2. Seon JY, Jeon WH, Bae SC, Eun BL, Choung JT, Oh IH. Characteristics in pediatric patients with coronavirus disease 2019 in Korea. J Korean Med Sci 2021;36:e148.
PUBMED | CROSSREF
3. Choi SH, Kim HW, Kang JM, Kim DH, Cho EY. Epidemiology and clinical features of coronavirus disease 2019 in children. Clin Exp Pediatr 2020;63:125-32.
PUBMED | CROSSREF
4. Wang L, Berger NA, Kaelber DC, Davis PB, Volkow ND, Xu R. Comparison of outcomes from COVID infection in pediatric and adult patients before and after the emergence of Omicron. medRxiv 2022. doi:
CROSSREF
5. Di Fusco M, Vaghela S, Moran MM, Lin J, Atwell JE, Malhotra D, et al. COVID-19-associated hospitalizations among children less than 12 years of age in the United States. J Med Econ 2022;25:334-46.
PUBMED | CROSSREF
6. Lee H, Choi S, Park JY, Jo DS, Choi UY, Lee H, et al. Analysis of critical COVID-19 cases among children in Korea. J Korean Med Sci 2022;37:e13.
PUBMED | CROSSREF
7. Molteni E, Sudre CH, Canas LS, Bhopal SS, Hughes RC, Antonelli M, et al. Illness duration and symptom profile in symptomatic UK school-aged children tested for SARS-CoV-2. Lancet Child Adolesc Health 2021;5:708-18.
PUBMED | CROSSREF
8. Nallasamy K, Angurana SK, Jayashree M, Mathew JL, Bansal A, Singh MP, et al. Clinical profile, hospital course and outcome of children with COVID-19. Indian J Pediatr 2021;88:979-84.
PUBMED | CROSSREF
9. Kwak JH, Lee SY, Choi JW; Korean Society of Kawasaki Disease. Clinical features, diagnosis, and outcomes of multisystem inflammatory syndrome in children associated with coronavirus disease 2019. Clin Exp Pediatr 2021;64:68-75.
PUBMED | CROSSREF
10. Feldstein LR, Tenforde MW, Friedman KG, Newhams M, Rose EB, Dapul H, et al. Characteristics and outcomes of US children and adolescents with multisystem inflammatory syndrome in children (MIS-C) compared with severe acute COVID-19. JAMA 2021;325:1074-87.
PUBMED | CROSSREF
11. Lee JK, Cho EY, Lee H. Multisystem inflammatory syndrome in children (MIS-C). Pediatr Infect Vaccine 2021;28:66-81.
CROSSREF
12. Gupta A, Madhavan MV, Sehgal K, Nair N, Mahajan S, Sehrawat TS, et al. Extrapulmonary manifestations of COVID-19. Nat Med 2020;26:1017-32.
PUBMED | CROSSREF

13. Ragab D, Salah Eldin H, Taeimah M, Khattab R, Salem R. The COVID-19 cytokine storm; what we know so far. Front Immunol 2020;11:1446.
PUBMED | CROSSREF

14. Manjili RH, Zarei M, Habibi M, Manjili MH. COVID-19 as an acute inflammatory disease. J Immunol 2020;205:12-9.
PUBMED | CROSSREF

15. Shah S, Meenakshisundaram R, Senthilkumaran S, Thirumalaikolundusubramanian P. COVID-19 in children: reasons for uneventful clinical course. Clin Exp Pediatr 2020;63:237-8.
PUBMED | CROSSREF

16. Choi JH, Choi SH, Yun KW. Risk factors for severe COVID-19 in children: a systematic review and meta-analysis. J Korean Med Sci 2022;37:e35.
PUBMED | CROSSREF

## 요약

코로나바이러스-19 (COVID-19)는 일반적으로 성인에 비해 소아에서 임상적으로 경한 양상을 보이며, 대부분의 소아에서 약 7일간의 격리가 끝나면 증상이 호전되어 특별한 이벤트 없이 퇴원하게 된다. 우리는 통상적인 임상경과와는 다르게, 격리 해제시점 또는 퇴원을 고려하고 있는 시점에 갑자기 악화된 임상증상을 보이는 3명의 청소년 환아들을 경험하였다. 세 명의 아이들은 공통적으로 신경학적 질환을 기저질환으로 가지고 있었다. Case 1은 울리히 선천성 근디스트로피로 진단받고 밤에만 이중양압기로 호흡보조를 받던 17세 남환의 경우로, 첫 COVID-19 증상 이후 9일이 지나고 증상 호전되어 퇴원을 준비하던 중 심폐정지가 발생하였다. Case 2는 뇌출혈 및 뇌경색 이후 병상에 누워 지내며 기관절개관 삽입 후 가정용 인공호흡기로 호흡보조를 받던 12세 여자 환아로, 증상이 호전되어 퇴원하였으나 첫 COVID-19 증상 이후 11일이 지난 후 심폐정지가 발생하였다. Case 3의 경우 조산아로 출생하여 뇌실출혈 및 수두증 진단받고 병상에 누워 지내나 호흡보조는 받지 않았던 12세 남자 환아로, 첫 증상 이후 호전추세였으나 10일 후 다기관 기능부전 확인되어 입원 진행하였다. 항바이러스제, 스테로이드제, 경험적 항생제가 투여되었고 중환자실 치료를 시행하였다. 세 환아들 중 2명 (case 1, 3)은 치료를 통해 호전되었으나, 1명 (case 2)는 심부전 진행하여 사망하였다. 이러한 경험에 비추어 볼 때, COVID-19 격리 기간이 끝나고 퇴원 가능한 시점이라도 갑작스러운 증상 악화를 보일 가능성이 있기에, 긴장을 놓지 않고 임상 증상의 변화를 확인하고 필요시 빠른 조치를 취해야 할 것으로 생각된다. 특히 신경학적 또는 호흡기적 만성 질환을 갖는 아이들에게 주의가 더 필요할 것으로 생각된다.