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Brief Analysis on Intravenous Immunoglobulin Treatment

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Abstract

Worldwide, the coronavirus 2 causing severe acute respiratory syndrome has infected and killed an alarming number of people. For the coronavirus illness of 2019 (COVID-19), no particular treatment has been internationally standardised; nevertheless, intravenous immunoglobulin (IVIG) has occasionally been utilised as adjuvant therapy in critically ill patients with COVID-19 pneumonia. We describe a case of a 50-year-old man who had severe COVID-19 pneumonia and had received an adjuvant 5-day IVIG regimen. We avoided using intrusive respiratory support. The patient made a full recovery and was released from the hospital without additional oxygen. Patients with severe COVID-19 pneumonia may have an improved chance of survival with a large IVIG dose. We looked at research on how IVIG use might help the early stages of the disease in the current report.

Keywords: Immunoglobulin; Coronavirus 2; Treatment; Clinical

Introduction

As of June 2021, the coronavirus disease 2019 (COVID-19) was caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which has infected more than 170 million individuals and killed more than 3.6 million people worldwide. As of June 2021, there has been confirmed cases of more over 379 000 COVID-19 cases in Panama, along with more than 6000 fatalities. For [1] COVID-19, no particular treatment has been widely standardised. Current treatment regimens were being tested in clinical settings at the time of this instance. An earlier SARS-CoV outbreak in 2002 employed the empirical delivery of intravenous immunoglobulin (IVIG) to patients with advanced illness. There was no control group; however it was thought that IVIG would be a useful treatment. With varying degrees of efficacy, IVIG has been utilised as an adjuvant therapy in critically ill COVID-19 patients. We describe a case of a patient with severe COVID-19 [2-5] pneumonia who successfully underwent a 5-day course of IVIG as adjuvant therapy and evaluated the research on the use of IVIG in COVID-19 patients. For the publication of patient data and photos, the patient gave written, informed consent. A 50-year-old man with a history of dyspnea and an oxygen saturation of 87% arrived to a hospital in Panama. Four days after returning from New Orleans, Louisiana, he began experiencing nasal congestion, coughing, sore throat, rhinorrhea, and fever with chills for a period of one week. He was exposed to patients who had flu-like symptoms and a fever while on his trip. The initial COVID-19 instances were noted at this period in LA and Panama.

Materials and Methods

Clinical course and treatment

Azithromycin 500 mg twice daily, prednisone 50 mg once daily, and budesonide/formoterol 160/4.5 g were the medications he was taking prior to being admitted to the hospital. He was given supplementary oxygen in the emergency room at a rate of 4 litres per minute (LPM) through a nasal cannula, but as soon as he was sent to the ward, his condition deteriorated and he needed up to 15 LPM through a reservoir mask. Bibasilar infiltrates could be seen on the initial chest radiograph, and a bilateral crazy-paving pattern could be seen on the computed tomography of the chest. He had a positive nasopharyngeal polymerase chain reaction test for SARS-CoV-2 (PCR). He began taking oral azithromycin, injectable ceftriaxone, and hydroxychloroquine. Despite receiving the best noninvasive oxygen therapy and lying in the prone position, he remained hypoxemic. D-dimers, ferritin, and C-reactive protein (CRP) levels were all significantly higher (Table 1). On day five of hospitalization, the patient was offered mechanical ventilation but declined. Then IVIG was started at 400 mg/kg/day for five days (total 25 g). Two days later the fever went away. He required 12 LPM of extra oxygen due to his chronic but less severe hypoxemia, and a chest x-ray showed infiltrates. A second round of antibiotics, including vancomycin and quinolones, was started because secondary bacterial pneumonia was thought to be the cause.

Resolution

On days 12 and 17 of hospitalisation, the SARS-CoV-2 nasopharyngeal PCR test was redone and revealed to be negative. After two weeks, the patient was gradually weaned off oxygen, and 18 days and mild but improving dyspnea later, she was discharged from the hospital.

Discussion

Under an electron microscope, the envelope of coronaviruses, which are positive-stranded RNA viruses that are encapsulated, resembles a crown. The cellular tropism of viruses depends on spikes, which are glycoproteins that impart viral identity and pathogenicity. The principal host cell receptor for the SARS-CoV-2 spike protein is angiotensin-converting enzyme 2, which is extensively expressed in [5-10] type II alveolar cells of the respiratory tract. This interaction could be the focus of a treatment strategy involving neutralising antibodies that could prevent viral fusion, receptor binding, and ultimately replication. Why some persons with no obvious risk factors experience severe disease is a subject of great curiosity. It's interesting to note that a genome-wide analysis carried out in Europe suggested that people with blood group A have a higher risk of developing serious respiratory illnesses. 5 Despite the fact that our patient had blood group A, this association was not known at the time and may or may not have

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Table 1: Hospitalisation day for the patient, necessary tests, and treatment.

DOH	1	3	4	5	6	7	8	9	10	11
Labs										
SARS-CoV-2 PCR	Detected									
CRP		37.58	143.75		141.02		86.04		19.53	
Ferritin				1945.1						5127.9

contributed significantly to the clinical course of our patient. The three phases of the clinical entity are thought to be the viremia phase (0–7 days), the acute phase of pneumonia (7–14 days), and the recovery phase (beyond 21 days). Critical immunological interaction is thought to take place during the pneumonial phase. The virus may be suppressed by a sufficient immune response, or there may be dysregulation resulting in a decrease in lymphocytes and an increase in pro-inflammatory cytokines and D-dimers. 6 By boosting the immune system, IVIG administration during this initial stage may be able to stop the progression. In our situation, IVIG was given shortly before the change from the acute to the pneumonia phase, and the result was acceptable. Its use in COVID-19 has been supported by the availability of IVIG among hospitals and its potential efficacy and safety in critically sick patients. Some individuals with severe COVID-19 pneumonia have improved survival after receiving a large dose of IVIG. A large dose of IVIG (25 g/day or 0.4 g/kg/day for 5 days) has been utilised in the majority of case reports, with successful results. 7–13 In a case series, a high IVIG dose helped stop the progression of pulmonary involvement, in contrast to one case where a lady received 5 g/day for 5 days before further deteriorating and needing extracorporeal membrane oxygenation. The timing of administration also appears to be important. One retrospective study found that when IVIG was administered within 48 hours of acute decompensation, ventilator use, hospital and intensive care unit lengths of stay, and mortality were all reduced in comparison to the previous 48 hours. 3 Despite showing no benefits of IVIG therapy in a randomised controlled study employing a new procedure, a lower dose, and a longer time of administration, it still supports safety because it did not reveal an increase in mortality. A relatively pure concentrate of polyclonal immunoglobulin G (IgG) generated from pooled human plasma from hundreds of donors is the main ingredient of IVIG. In replacement therapy, the pathogen is neutralised, toxic substances are rendered inactive, toxins are opsonized, B and T cell activity is increased, and complement is activated. The neutralisation of autoantibodies and pro-inflammatory cytokines, inhibition of activated complement and/or adhesion molecules, disruption of the idiotypic/anti-idiotypic network, and improved autoantibody clearance are only a few examples of anti-inflammatory actions. 19 If any of these functions apply to coronaviruses, such as SARS-CoV-2, more research is required. It has been hypothesised that IVIGs can treat COVID-19 through a number of different methods. The formation of primed antibodies against prior coronavirus infection, or antibody-dependent enhancement, has been suggested as a potential mechanism to explain differences in severity between nations. 20 Unaltered human IVIGs sold commercially have shown some in vitro cross-reactivity to SARS-CoV-2 and other coronaviruses. The tested preparations came from donors in European and American nations. IVIG preparations have been found to contain antibody reactivity against the SARS-CoV-2 S1 protein. 21 A hopeful study showed a quick rise in the concentration of particular neutralising antibodies among preparations created during the pandemic year, despite the fact that a subsequent investigation found no cross-neutralization antibodies Additional research is still needed to determine how neutralising antibodies act, and other potential mechanisms, like anti-inflammatory effects, may also be at play. It has been suggested, but not yet shown, that the inflammatory cascade can be improved by binding cytokines and other antibodies, complement scavenging, inhibiting innate immune cells and effector T-cell activation, and increasing Tregs. Undoubtedly, immunisation would be the most effective strategy to lower transmission and the likelihood of severe cases. However, when the condition arises in persons who run the danger of advancing to severe COVID-19 pneumonia, substantial doses of IVIG may still be helpful.

Conclusion

In conclusion, this case report showed that individuals with severe COVID-19 pneumonia may benefit from receiving a high dosage of IVIG early on. The functionality, efficacy, and advantages of IVIG in patients with severe SARS-CoV-2 infection, however, remain largely unknown.

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Declaration of conflicting interests

No potential conflicts of interest were disclosed by the author(s) with regard to the research, writing, or publication of this paper.

Ethical approval

For reporting individual instances or case series, our institution does not require ethical approval.

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