POSITIVE OUTCOME IN A PATIENT WITH COVID-19 AND MYSTHENIA GRAVIS AFTER INTRAVENOUS IMMUNOGLOBULIN

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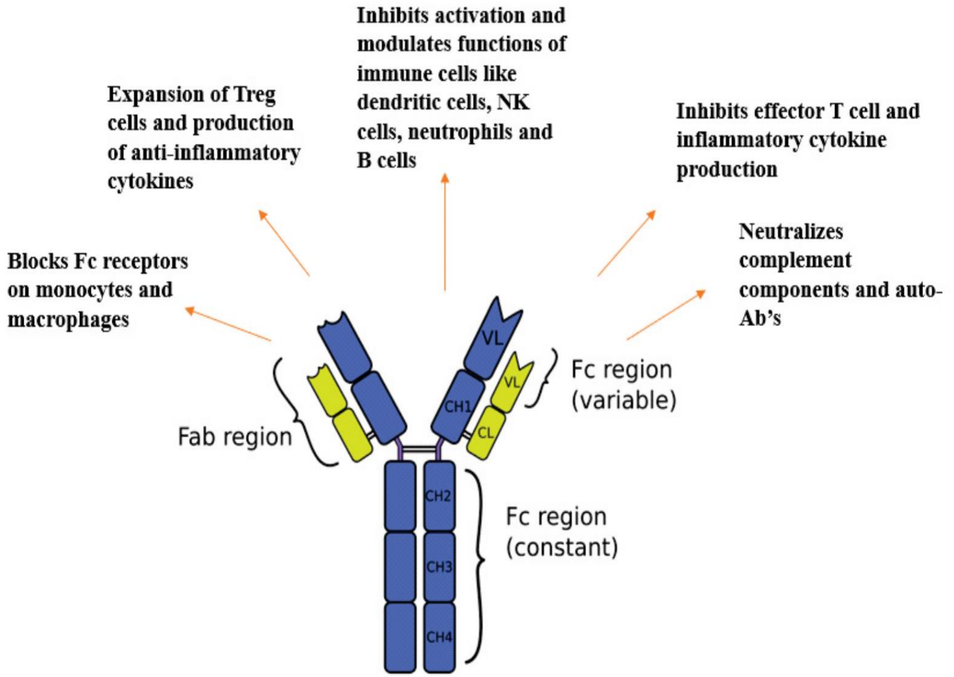
BACKGROUND

Viral infections can exacerbate or precipitate neurological disorders by inducing fever, causing immune dysregulation and triggering autoimmunity. Similarly, risk factors like muscular weakness of the chest or diaphragm, poor airway clearance from weak cough, cardiac involvement, immune suppressive therapies and coexisting comorbidities in patients with neuromuscular disorders could potentially result in severe infections.

We report a patient with history of acetylcholine receptor antibody positive myasthenia gravis who presented with acute worsening of her myasthenic symptoms and COVID-19 infection was later found during evaluation as a possible trigger.

CASE PRESENTATION

HPI:
A 64-year-old female presented to the hospital with generalized muscular weakness and lethargy.
PMH:
Hypertension, diabetes mellitus, myasthenia gravis on pyridostigmine and scheduled IVIG infusions.
Physical exam:
Vitals- respiratory Rate-26/min, pulse oximetry-85% on room air.
Bilateral ptosis, difficulty holding head up, motor strength 1/5 in all four extremities.
Laboratory:
White cell count- 26.4 10*3/ul with lymphocytopenia, ferritin-405.4 ng/ml, C-reactive protein-144 mg/L, aspartate aminotransferase-187 U/L, alanine aminotransferase-104 U/L, SARS-CoV2 RT-PCR- positive.
Imaging:
Chest x-ray- patchy bilateral airspace opacities.
Hospital course:
Patient was placed on BiPAP (Bilevel positive airway pressure) to maintain oxygen saturation above 92%. IVIG 400 mg/kg body weight for 5 days, pyridostigmine 60 mg every six hours, methylprednisolone 40 mg IV every 8 hours. and remdesivir 100 mg IV daily for 5 days were initiated. Patient's clinical status including muscle weakness improved, and oxygen supplementation was gradually titrated down to room air. Patient was discharged home after 11 days of hospitalization.



DISCUSSION

IVIG contains antibodies from the plasma of thousands of donors (unlike convalescent plasma which is derived specifically from patients who have recovered from COVID-19 infection). It contains about 95% IgG and trace amounts of IgA and IgM.

Mechanism of action of IVIG:
1)IV immunoglobulins modulate the immune system by acting on multiple components of the innate and adaptive immunity.

2) Commercially available IVIG preparations contain antibodies against the human endemic coronaviruses that cause common cold. Because of the morphological similarities between coronaviruses that cause mild endemic (HCoV-OC43, HCoV-HKU1, HCoV-229E, and HCoV-NL63) and severe epidemic infections (SARS-CoV-2), it has been hypothesized that neutralizing antibodies against endemic coronavirus antigens found in these products might cross react against the epidemic coronavirus. ELISA based assays have shown up to 90% cross neutralizing capacity of these antibodies against SARS-CoV2 virus.

CONCLUSION

At least in our patient IVIG therapy through its impact on the innate and adaptive immune systems appears to have helped her recovery from both the myasthenia gravis exacerbation and COVID-19 despite multiple high-risk comorbidities.

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