TABLE 2. *Laboratory and radiological parameters*

Parameter		Normal Value	Patient 1	Patient 2	Patient 3
Hemoglobin (gm/dl)		13-17	12.5	13.2	14.1
White cell count (per cmm)		4,000-11,000	8200	5000	27,300
Neutrophils (%)		40-70	81	87	94
Lymphocytes (%)		20-45	11	9	4
Monocytes (%)		2-10	8	4	2
Eosinophils (%)		1-6	0	0	0
Basophills (%)		0-1	0	0	0
Platelet count (no./cmm)		1.5-4.5	4.28	1.67	1.24
Mean corpuscular volume (fL)		80-96	89.6	88.7	87.2
Mean corpuscular hemoglobin (pg)		27-34	28.2	28.8	27.5
Creatinine (mg/dl)		0.7-1.2	1.05	0.87	1.1
Blood urea (mg/dl)		15-40	35	41	142
Prothrombin time (seconds)		14	14	14	16
INR			1.0	1.0	1.1
Activated partial-thromboplastin time (seconds)		28-32	30	28	32
D-Dimer (mg/dl)		< 250	398.3	345	Ø1000
Serum ferritin (ng/ml)		10-291	459.1	281.9	721.0
Lactate Dehydrogenase (U/L)		313-618	427	346	372
ESR (mm/hour)		0-9	28	19	30
C-RP (mg/dl)		<0.6	4.8	2.4	4.8
IL-6 (pg/ml)		0-6.4	44.27	62.34	37.10
HbA1c (%)		3-6	5.4	7	6.34
CT Chest	Percentage lung involvement		25-30%	80-85%	
	CORADS		6	6	
	CT severity score		9/25	18/25	

of symptoms and needed high flow oxygen via non re-breathing mask for maintaining arterial oxygen saturation. The patient deteriorated rapidly while being hospitalized, developed hypoxemic respiratory failure and was administered intravenous Immunoglobulin at the dose of 400mg/kg/day for 3 days and subsequently succumbed to the disease. He was intubated and on mechanical ventilatory support on the day of death. He had no exacerbation of peripheral myasthenia syndromes like ptosis, extra-ocular muscle weakness, bulbar or limb weakness during hospitalization.

The clinical course of these patients was observed during hospitalization and the details of the same are mentioned in table 1. The laboratory and CT chest findings of the patients are mentioned in table 2.

None of the patients had an exacerbation of peripheral myasthenia syndromes like ptosis, extra-ocular muscle weakness, bulbar or limb weakness during hospitalization. Therapy for MG was not altered during hospitalization for any of the patients.

Depending on the symptoms, level of the serum inflammatory markers and CT chest findings, specialized therapies were administered to the patients. There was no relation of ultimate patient outcome with the duration of MG symptoms, previous therapy of MG, and the pre-existing comorbidities. Further, the outcome of these patients depends on the levels of serum inflammatory markers, percentage of lung involvement and CT severity score for SARS-CoV-2.

DISCUSSION

Infections are a known common trigger for myasthenic exacerbations [4]. Theoretically, there is a higher risk of experiencing severe manifestations of SARS-CoV-2 due to the common use of immunosuppressive drugs and potential respiratory failure in relation to respiratory muscle weakness. Additionally, experimental therapies for SARS-CoV-2 like azithromycin and hydroxychloroquine may also trigger a myasthenic exacerbation [5,6].

In our patients, we had no exacerbation of myasthenic symptoms which is a very noteworthy ob-

servation, as respiratory infections are known to be the most common precipitant of respiratory failure and crisis in myasthenia patients [7].

Previous use of steroids plus immunosuppressive therapies did not seem to determine an unfavorable outcome in our patients. Though immuno-suppressed patients could be at a higher risk for a more severe COVID-19 course, growing evidence shows that immunosuppression might play a protective role, reducing the immune response that leads to an inflammatory cytokine storm and to clinical deterioration [8,9].

CONCLUSIONS

Our series is limited by the small number of patients and short duration of follow-up, which did not include the postinfectious period, a time of risk for myasthenic exacerbation. Larger and longer observations are needed to more fully understand: whether patients with MG face special risks from SARS-CoV-2 infection or treatments, whether baseline therapies impact risk and best principals for management and treatment of SARS-CoV-2 in patients with MG.

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