Increased rates of idiopathic inflammatory myopathies during the COVID-19 pandemic: a single-centre experience

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Abstract

Objective

Higher-level evidence is required to discern whether the incidence of idiopathic inflammatory myopathies (IIM) has increased during the COVID-19 pandemic and whether the disease pattern and course have changed. We aimed to analyse patients who were diagnosed with IIM at our tertiary care centre during the pandemic and compare them with IIM patients diagnosed before COVID-19.

Methods

We retrospectively analysed the medical records of adult patients (>18 years) who were diagnosed with IIM during COVID-19 versus a control group of patients diagnosed before the outbreak. Included were patients whose diagnosis was made at the Department of Medicine and Rheumatology Unit of Hadassah Medical Center, Jerusalem, Israel. We also conducted a comprehensive review of the literature regarding SARS-CoV-2 infection and vaccine-induced IIM.

Results

Our study yielded 18 and 16 diagnosed IIM patients over periods of 27 and 56 months in the COVID-19 and pre-pandemic cohorts, respectively. These constitute incidence rates of 0.66 and 0.28 patients/month, respectively, marking an increased rate in the COVID-19 group. Unique features were noted in IIM patients who were diagnosed during the pandemic. This includes male predominance (M:F ratio of 12:6), higher hospitalisation rate (0.77 vs. 0.43 admitted/total patients) and increased number of patients with CPK >10,000 U/L (3 vs. 1 patient). Despite the more severe presentation and course in the pandemic group, survival was comparable between the groups.

Conclusion

The incidence of IIM increased during the COVID-19 pandemic. These patients display unique features and a more severe presentation. Fortunately, the prognosis remains unchanged.

Key words

COVID-19, infection, vaccination, idiopathic inflammatory myopathies

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Introduction

Infectious diseases have long been linked to the development or exacerbation of autoimmune disorders (AIDs). Group A streptococcal (GAS) infection is considered a hallmark of infection-induced autoimmunity due to the molecular similarities between GAS M protein and myocytes and the consequent development of rheumatic fever (1). This infection-autoimmunity correlation can be found for other pathogens as well, although a protective role of infectious agents can also be seen, as in the case of hepatitis B virus and systemic lupus erythematosus (1).

The Coronavirus disease 2019 (COVID-19) pandemic had a notable influence on different fields of medical practice and health care delivery, including rheumatology and immunology. This might have been augmented by the contribution of the different vaccinations against Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) (2-4). During the pandemic, reports of the induction and exacerbation of AID by SARS-CoV-2 have been accumulating. Our group recently reported a single-centre experience of AID induction by SARS-CoV-2 infection (5). This was supported by various other studies, including reports of COVID-19 inducing Guillain-Barré syndrome, multisystem inflammatory syndrome in children (MIS-C), and other diseases (6). SARS-CoV-2 mRNA vaccines were also postulated to induce AID, as in the case of SARS-CoV-2 vaccine-induced myocarditis (7).

Idiopathic inflammatory myopathies (IIM) are systemic AIDs characterised by myositis and possible inflammation of the skin and visceral organs. Different disease subtypes are defined according to clinical, serologic, and pathologic findings. The aetiology is multifactorial, but infections and vaccinations are thought to play a role (8). Studies regarding SARS-CoV-2-induced IIM are lacking. Review of the literature yields mostly single case reports (9-20) , with larger case series referring to SARS-CoV-2 vaccines (21, 22). Certainly, higher-level evidence is still required to discern whether the incidence of IIM has increased during the pandemic and whether the disease pattern and course have changed.

Here, we aimed to analyse and characterise patients who were diagnosed with IIM at our tertiary care centre during the pandemic and compare them with IIM patients diagnosed before COVID-19.

Methods

Study design

We conducted a retrospective analysis of medical records of adult patients (>18 years) who were diagnosed with IIM during the COVID-19 pandemic *versus* a control group of patients diagnosed before the outbreak. Patients included were those whose diagnosis was made at the Department of Medicine and Rheumatology Unit of Hadassah Medical Center, Jerusalem, Israel. Analysis included admitted as well as outpatients. We evaluated patient characteristics, clinical presentation, laboratory parameters, treatment, and outcome.

Inclusion and exclusion criteria

Patients were included if they met the European Alliance of Associations for Rheumatology (EULAR)/ American College of Rheumatology (ACR) classification criteria for adult IIM (23). The disease subgroups were classified as suggested by Dalakas *et al*. (24). Anti-synthetase syndrome was considered a distinct entity. IIM patients who lacked data in their medical records were excluded from the study.

Review of the literature

We further conducted a review of the English-language literature regarding IIM induced by SARS-CoV-2 infection or vaccine. The search was defined by the key words "myositis" or "idiopathic myopathy" and "COVID-19 infection" or "COVID-19 vaccine". The review was limited to publications published from July to December 2022.

Ethics review

The study was approved by Hadassah's institutional review board (IRB, HMO-0322-22). The IRB granted a waiver from gathering informed consent from the patients.

Results

Patient characteristics

Results are presented in Table I. Our study yielded 18 and 16 diagnosed IIM patients over periods of 27 and 56 months, in the COVID-19 and pre-pandemic cohorts, respectively. This constitutes incidence rates of 0.66 and 0.28 patients/month, respectively, marking an increased rate in the COVID-19 group. Interestingly, although female predominance is usually reported in IIM, male predominance was observed in the COVID-19 cohort (M:F ratio of 12:6), as compared with the female predominance before the pandemic (M:F ratio of 5:11). Furthermore, the COVID-19 cohort was characterised by over-representation of the Jewish population (89%), as well as a slightly older age (mean age 64.0 *vs*. 55.4 years) upon disease presentation.

Clear documentation of SARS-CoV-2 infection in the COVID-19 cohort was not available, due to the mass infection rate at the beginning of the pandemic. However, data regarding SARS-CoV-2 vaccine schedules were precise, and only 5 of these patients presented with IIM prior to SARS-CoV-2 vaccine administration.

Clinical and laboratory presentation

Muscle biopsies were available in 12 and 15 patients in the pre-COVID-19 and pandemic groups, respectively. Magnetic resonance imaging (MRI) was performed in almost all patients. Considering clinical presentation, myositis-specific antibodies, and biopsies, diagnosed IIM consisted of polymyositis (PM), anti-synthetase syndrome, dermatomyositis (DM), overlap myositis, and immune-mediated necrotising myopathy (IMNM). PM and DM constituted most of the patients in each cohort, with comparable numbers of patients (n=6 *vs*. 7 [PM] and n=3, each [DM], in the COVID-19 and pre-pandemic cohorts, respectively). However, over-representation of IMNM can be seen in the COVID-19 cohort as compared with the pre-pandemic cohort (3 *vs*. 1 patient, respectively). Serum C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) were slightly higher in the COVID-19 cohort, although not statisti-

Table I. Summary of patients with immune-mediated myositis before and during COVID-19 pandemic.

Parameter		Pre-COVID-19 pandemic	During COVID-19 pandemic
Number of patients		16	18
Follow-up period (months)		56	27
Prevalence (patients/month)		0.28	0.66
Diagnosis	PM	7	6
	Anti-synthetase syndrome	2	1
	DM	3	3
	IMNM	3	6
	Overlap myositis	*1	2**
Mean age (range; years)		55.4 (46-76)	64.0 (56-76)
Gender (male: female)		5:11	12:6
Ethnicity (Jews: Arabs)		9:7	16:2
Hospital admission rate (admitted/total patients)		0.43	0.77
Diagnosis of co-existing AID		1	4
Statins treatment before myositis		4	7
Clinical manifestations	Joint disease	7	5
	ILD	5	1
	Cardiac	3	4
	Dysphagia	2	2
	Raynaud	4	2
Inflammatory markers	Mean CRP (range; <0.5 mg/dL)	3.9 (0-17)	8.0 (0.05-20.2)
	Mean ESR (range; <10 mm/H)	35.7 (6-82)	49.5 (14-98)
Number of patients with CPK >10,000 U/L		1	3
Number of patients with serum myositis-associated or specific autoantibodies		10***	11+
Serum autoantibodies	ANA	7	7
	Anti-RO	3	2
	Anti-La	0	1
	Anti-JO-1	5	2
	Anti-HMG-CoA reductase	1	2
	Anti-TIF-1γ	0	1
	Anti-NXP-2	0	1
	Anti-SAE-1	0	1
	Anti-PM-SCL	0	2
	Anti-SRP	0	1
Muscle biopsy		12++	15 +++
Treatment	GCS	16	1
	MTX	11	8
	Azathioprine	3	2
	Hydroxychloroquine	0	1
	IVIG	2	3
	Rituximab	2	2
	MMF	2	0
Outcome (N of survival)		15	17

PM: polymositis; DM: dermatomyositis; IMNM: immune-mediated necrotising myositis; SS: systemic sclerosis; AID: autoimmune diseases; ILD: interstitial lung disease; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; ANA: anti-nuclear antibody; CPK: creatine phosphokinase; GCS: glucocorticosteroids; MTX: methotrexate; IVIG: intravenous immunoglobulins; MMF: mycophenolate mofetil.

* Overlap with mix connective tissue disease.

** Overlap with systemic sclerosis and Sjögren's syndrome.

++Two patients did not undergo muscle biopsy and had anti-synthetase syndrome. One patient had a suggestive skin biopsy.

+++One patient with amyopathic DM did not undergo muscle biopsy. However, he had suggestive findings on skin biopsy.

Table II. Review of previously reported patients with idiopathic inflammatory myopathies induced by SARS-CoV-2 infection or vaccine.

Number of patients	Age on IIM diagnosis (years)	Gender	Underlying statin treatment	IIM subtype	Symptoms onset from SARS-CoV-2 inducer (months)	Induction of IIM by SARS-CoV-2 infection (number of patients)	Induction of IIM by SARS-CoV-2 vaccine (number of patients)	Supporting muscle biopsy (number of patients)	ANA positivity (number of patients)	Myositis-specific or associated autoantibodies positivity (number of patients)	Elevated CPK levels (number of patients with CPK>10000 U/mL)	Hospitalisation rate (admitted/ diagnosed patients)	Treatment/outcome	Follow up period (months)	Reference
5	36-79	F	-	DM	1-30 days	-	+(5)	1	3	4	-	1/3	GCS, MMF, IVIG / CR	NA	(21)
1	54	F	+	SIBM	3	+ (1)	-	1	1	1	+ (0)	0/1	NSAID/ CR	24	(9)
1	58	M	-	PM	6	+ (1)	-	0	0	0	+ (0)	0/1	GCS/ CR	NA	(10)
1	67	M	-	necrotizing autoimmune myositis	NA	+ (1)	-	1	0	1	+ (1)	1/1	GCS/ CR	15	(11)
1	66	F	-	IBM	2 weeks	+ (1)	-	1	1	1	+ (0)	0/1	GCS/ CR	NA	(12)
1	47	M	-	NA	3	+ (1)	-	0	NA	NA	+ (0)	0/1	NSAID, GCS/ CR	1	(13)
1	47	M	-	DM	5	-	+ (1)	1	1	1	+ (0)	1/1	GCS, IVIG/ CR	3	(14)
1	64	M	-	Delayed Localized Necrotizing Inflammatory Myositis	8	-	+ (1)	1	0	0	-	1/1	GCS/ CR	NA	(15)
1	64	F	-	NA	15 days	-	+ (1)	0	NA	NA	NA	1/1	NA/ CR	4	(16)
1	35	M	-	DM	2	+ (1)	+ (1)	1	1	1	+ (1)	1/1	GCS/ CR	1	(17)
1	53	M	-	NA	11 days	-	+ (1)	NA	NA	NA	+ (0)	0/1	NSAID/ CR	5 weeks	(18)
1	36	M	-	PM	6 days	-	+ (1)	1	NA	NA	+ (0)	1/1	GCS/CR	NA	(19)
1	89	M	-	NA	NA	+ (1)	-	NA	NA	NA	+ (0)	1/1	GCS/ CR	10 days	(20)
4	19-57	3F 1 M	-	DM	1-7 days (vaccine) 2 weeks (infection)	+ (1)	+ (3)	NA	0	2	+ (0)	NA	GCS/ CR	NA	(22)

M: male; F: female; IIM: idiopathic inflammatory myopathies; DM: dermatomyositis; SIBM: sporadic inclusion body myositis; PM: polymyositis; CPK: creatine phosphokinase; ANA: anti-nuclear antibody; NA: data is not available; GCS: glucocorticosteroids; IVIG: intravenous immunoglobulins; NSAID: non-steroidal anti-inflammatory drugs; MMF: mycophenolate mofetil; CR: complete resolution of symptoms.

cally significantly (*p*-values of 0.17 and 0.2, respectively). Anti-Jo-1 antibodies were more frequent in the pre-COVID-19 cohort as compared with IIM patients diagnosed during the pandemic (5 *vs*. 2 patients, respectively). No major differences in the pattern of myositis-specific or associated autoantibodies between the groups were observed. Anti-nuclear antibody (ANA) was most prevalent (n=7 in each group).

Hospitalisation rate upon diagnosis, as an indicator of disease severity, was higher in the COVID-19 cohort (0.77 *vs*. 0.43 admitted/total patients). This is further supported by the increased number of patients with serum creatine phosphokinase (CPK) levels >10,000 U/L in the COVID-19 group as compared with the pre-pandemic cohort (3 *vs*. 1 patient).

Treatment and outcome

Treatment was similar between the groups (Table I). It consisted of glucocorticosteroids (GCSs) and methotrexate in most patients. Other agents used were azathioprine, mycophenolate mofetil, and hydroxychloroquine. Two patients in each group received biological treatment with rituximab. Patient outcomes were also similar, with comparable survival rates (15/16 and 17/18 of the patients in the pre-COVID-19 and pandemic cohorts, respectively).

Review of the literature

Our review of the literature is summarised in Table II. Our search yielded 7 and 13 patients with IIM induced by SARS-CoV-2 infection and vaccination, respectively. One patient was reported to have both SARS-CoV-2 infection and vaccine as possible triggers. Six and 8 patients, respectively, in the infection- and vaccine- induced IIM patients were males, which indicates a male predominance in both conditions. Ages varied, ranging between 19 and 89 years. IIM subtypes consisted of DM (n=11), PM (n=2), inclusion body myositis (n=2), and undetermined IIM subtype (n=6). ANA positivity was noted in 7 patients. CPK >10000 U/L was noted in only 2 patients, both with infection-induced IIM. Myositis-specific autoantibodies were noted in 11 patients. Muscle biopsies were done in only 7 patients. Other patients were reported to be diagnosed by clinical presentation, laboratory workup, and MRI studies. GCSs were the preferred treatment in the reported patients (n=18). Complete resolution of symptoms and favourable outcome were noted in all reported patients.

Discussion

In this study, we analysed IIM patients diagnosed at our tertiary care centre before and during the COVID-19 pandemic. Our report demonstrates the male predominance of patients diagnosed with IIM, mostly PM and DM, during the pandemic, as compared with the female predominance in the pre-COVID-19 cohort, the latter being compatible with the literature (25). This male predominance may represent a unique feature, in concordance with that seen in

SARS-CoV-2 mRNA vaccine-induced myocarditis (7). Our results also agree with the male predominance seen in previously reported SARS-CoV-2 infection–induced IIM (Table II).

Our analysis also identified an increased rate of IMNM during the COVID-19 pandemic. This could be related to the infection or vaccination serving as a "second hit" in those patients predisposed to IMNM due to statin use. Despite the low number of patients, the rate of anti-synthetase syndrome was not increased in the COVID-19 cohort, contrary to previous speculations (26).

An additional interesting finding was over-representation of the Jewish population among IIM patients in the COVID-19 group. Arabs constitute about 38% of the population in Jerusalem, and the ~44% (7/16) representation of Arab patients in the pre-COVID-19 cohort is consistent with this. On the other hand, the percentage of Arab patients with IIM in the COVID-19 cohort was ~11%.

It is noteworthy that anti-Jo-1 positivity tended to be more frequent in the pre-COVID-19 group. The presence of this antibody, however, did not necessarily translate to significant differences in the occurrence of the anti-synthetase syndrome. The latter was present in 2 patients in the pre-COVID-19 cohort and in 1 patient during the pandemic. No major difference in the pattern of myositis-associated antibodies was present between the two groups. While myositis-specific antibodies were tested in most patients post COVID-19, this was not the case in the pre-COVID-19 cohort, excluding anti-Jo-1, thus limiting the ability to draw conclusions in that regard.

Concerning IIM clinical severity, different indicators are shown in our study to support the notion of a more severe course during COVID-19. This includes a higher hospitalisation rate, an increased number of patients with CPK >10,000 U/L, and slightly but not statistically significantly increased levels of serum inflammatory markers, which are usually known not to be markedly elevated in IIM (25). In this regard, it is worth mentioning that COVID-19 tends to run a more severe course in males (27), and male predominance was observed our COVID-19 cohort. IIM severity, as indicated by serum CPK>10000 U/L, was seen in 2 previously reported patients, both with infection-induced myopathy (Table II). The outcomes of IIM patients, however, were comparable between the two groups in our study and with previously published reports of patients with vaccine- and infection-induced IIM. This supports the notion of a favourable outcome in these patients.

Different mechanisms have been suggested to explain infection- or vaccine-induced AID, including a bystander effect, molecular mimicry, and viral persistence (6). Direct SARS-CoV-2 infection of myocytes, via angiotensin-converting enzyme (ACE)-2, was previously suggested as a possible explanation for COVID-19-associated IIM (28). Increased ACE-2 expression via activation of toll-like receptor (TLR)-4 has also been proposed (28). Other postulated mechanisms include an increase in pro-inflammatory cytokines, an autohumoral response against myocytes, and T-cell clonal expansion (28).

Our report has several limitations. It is retrospective and includes a small number of patients. Solid conclusions regarding myositis-specific antibody comparisons are limited, due to the reduced numbers of panels performed in the pre-COVID-19 cohort (excluding anti-Jo-1, done in all patients). Moreover, it is difficult to know whether the increased rate of IIM during the pandemic in our study is related to SARS-CoV-2 infections or vaccines, as most patients in this group were vaccinated during this period and no precise documentation of their SARS-CoV-2 infection is available. However, the increased rates of IIM during COVID-19, the higher proportion of IMNM, the male predominance, and the elevated rates of hospital admissions upon diagnosis are important characteristics that emphasise the unique autoimmune features of patients during the pandemic.

In conclusion, we have demonstrated increased rates of IIM in patients diagnosed during the COVID-19 pandemic. These patients display unique features and a more severe presentation. Fortunately, the prognosis remains unchanged.

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