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Thyroid disorders in idiopathic inflammatory myopathies: prevalence and related clinical scenarios

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Abstract

Background Little is known about the relationship between thyroid diseases (TDs) and Idiopathic inflammatory myopathies (IIMs). This study aimed at evaluating the prevalence of TDs in a monocentric cohort of patients with IIMs, exploring possible correlations with clinical phenotype, organ involvement, comorbidities and quality of life (QoL).

Methods We retrospectively analysed medical records of patients with IIM according to the EULAR/ACR 2017 criteria, collecting data about demography, IIM subset, disease duration, autoantibody profile, organ involvement and comorbidities. Besides, we registered the occurrence of Hashimoto Thyroiditis (HT), Multinodular goitre (MNG), Grave's Disease (GD) and Thyroid papillary cancer (TPC). In addition, some different patient reported outcomes were administered, to collect data on QoL.

Results A total of 191 patients were enrolled: 125 (65.4%) were female, with a mean age of 66.6 ± 13.5 years and a mean disease duration of 9.8 ± 7.4 years; 111 (58.1%) had dermatomyositis (DM), 50 (26.2%) polymyositis (PM), 11 (5.8%) clinically amyopathic DM or inclusion body myositis, 6 (3.1%) immune-mediated necrotizing myopathy and 2 (1%) juvenile DM. Ninety-nine patients (51.8%) had a TD; 53/191 (27.7%) had MNG, 42/191 (22%) had HT and 3/191 (1.6%) GD. One patient (0.5%) had a TPC. The presence of a TD was associated with oesophagus' involvement and with a higher risk of osteoporosis and fragility fractures ($p \leq 0.01$). Moreover, patients with TDs tended to accumulate a greater number of comorbidities ($p < 0.001$) and showed higher values of cumulative dose of glucocorticoids ($p = 0.032$). Finally, considering patients' QoL, the presence of a TD was associated with lower values of the SF-36 bodily pain domain ($p = 0.006$).

Conclusions More than half of our IIMs patients had a TD, with a higher prevalence of both MNG and HT. Our results partially fill a lack of knowledge about the relationship between TDs and IIMs, stressing the opportunity of regularly screening patients for thyroid function.

Clinical trial number Not applicable.

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Keywords Idiopathic inflammatory myopathies, Thyroid diseases, Quality of life, Dysphagia, Pain perception, Disease activity, Disease damage

Introduction

Thyroid diseases (TDs) are relatively common endocrine disorders in the general population (GP) [1]. In particular, Hashimoto Thyroiditis (HT), Multinodular Goitre (MNG), Grave's Disease (GD), Papillary thyroid cancer (TPC) prevalences in GP correspond respectively to about 12%, 10%, 1.3% and 0.2% [2–5].

TDs are, also, a well-known comorbidity of patients with autoimmune systemic diseases, such as systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA) [6] and might compromise the health status of patients, owing to their possible impact on cardiovascular (CV) risk, bone mineral density (BMD) and muscle function.

However, the evidence of a link between thyroid disorders and Idiopathic Inflammatory Myopathies (IIMs) is scarce, although both hyper- and hypothyroidism have been reported [7]. IIMs are a heterogeneous group of chronic autoimmune diseases, sharing symptoms of muscle weakness due to muscle tissue inflammation. Other organs, in particular skin, lungs, upper digestive tract and heart, can be frequently involved by systemic inflammation [8]. As a result of both disease activity, drug toxicity and damage accrual, IIM patients tend to have an impaired quality of life (QoL) [9].

The endocrine system might influence several aspects of muscular functionality, with possible consequences on IIMs' disease course: in particular, thyroid hormones play a pivotal role in controlling myogenesis, damage repair and also transcription profile of muscle fibres [7]. Moreover, TDs represent an established cause of high bone turnover with an accelerated bone loss, leading to osteoporosis (OP) and increased susceptibility to fragility fractures (FFx) [10, 11]. Finally, TDs might influence the CV system via plenty mechanisms, including an increased risk of dyslipidaemia, hypertension, systolic and diastolic myocardial dysfunction, as well as endothelial dysfunction [12], thus potentially amplifying CV damage accrual in IIM patients [13].

The aim of this study was to evaluate the prevalence of TDs in a monocentric cohort of patients with IIMs, exploring possible correlations with autoantibody profile, organ involvement, comorbidities and patients' QoL.

Methods

We retrospectively analysed medical records of patients diagnosed with IIM followed at our specialistic Myositis Clinic (University medical centre of Pisa) from January 2021 to March 2024. Our monocentric cohort included patients with a diagnosis of IIM fulfilling the EULAR/ACR 2017 classification criteria [14].

Patients under 18 years and those who did not sign the informed consent form were excluded.

The following data were collected from clinical charts: demography, subset, disease duration and autoantibody profile, comorbidities (hypertension, atrial fibrillation, dyslipidaemia, diabetes, obesity, OP, FFx, cataract, dysthymia, chronic renal failure, fibromyalgia, neoplasms, hyperuricemia, gout, myocardial infarction, stroke), family history of autoimmune disease, other autoimmune disease, smoking status, obesity and social-economic status. We also collected data about organ involvement (muscle, oesophagus, skin, lung, heart, joints, microcirculation). We considered the presence of an oesophageal involvement in those cases of clinical or subclinical dysphagia [highlighted by an oesophagus' X-Ray or an oropharyngoesophageal scintigraphy (OPES)]. We considered the presence of heart involvement in those cases of inflammatory cardiomyopathy.

In addition, just before the visit, all the patients were asked to fill in the following patient reported outcome measures (PROMs) to evaluate QoL's outcomes: Short-Form 36 (SF36) [15], Health Assessment questionnaire (HAQ) [16], Hospital Anxiety and Depression Subscales (HADS) [17] and Functional Assessment of Chronic Illness Therapy – Fatigue (FACIT-F) [18].

Patients' serology was determined by line blot immunoassay (EUROLINE Autoimmune Inflammatory Myopathies 16 Ag, Euroimmun, Lubeck, Germany).

The occurrence of HT, MNG, GD and TPC were also evaluated systematically in all patients. In particular, the diagnosis of a TD was collected during the anamnesis. In the same context, if available, thyroid function tests [thyroxine (T4), triiodothyronine (T3), thyroid-stimulating hormone (TSH), anti-thyroperoxidase (TPO), anty-thyreoglobulin (TG) and TSH receptor antibodies (TRAb)], ultrasound data and any specific therapies were collected. Otherwise, we prescribed the dosage of hormones, the research of autoantibodies and an ultrasound control. In patients without a diagnosis of TD we prescribed TSH and autoantibodies testing. In cases of altered tests or a palpable thyroid during the physical exam, we prescribed an ultrasound examination.

The study was planned under the Declaration of Helsinki, and it received local ethics committee approval (Comitato Etico di Area Vasta Nord Ovest), and the committee's reference number is 20,070.

Statistical analysis

Both univariate (Chi-square, t-test and ANOVA) and multivariate analyses (linear regression) were assessed.

Table 1 Epidemiological and clinical characteristics of patients

N° of patients	191
Male/Female	66/125
Mean age (year ± SD)	66.6 ± 13.5
Mean disease duration (year ± SD)	9.8 ± 7.4
Muscle involvement	161/191 (84.3%)
Esophageal Involvement	114/191 (59.7%)
Skin Involvement	100/191 (52.4%)
Raynaud Phenomenon	83/191 (43.5%)
Lung Involvement	78/191 (40.8%)
Articular Involvement	47/191 (24.6%)
Cardiac Involvement	12/191 (6.3%)

Table 2 MSA and MAA distribution in our cohort

Anti-Ro52 (%)	55 (28.8)
Anti-Mi2 (%)	25 (13.1)
Anti-PM-Scl (%)	23 (12)
Anti-Jo1 (%)	17 (9)
Anti-TIF1γ (%)	15 (7.9)
Anti-SRP (%)	11 (5.7)
Anti-MDA5 (%)	10 (5.2)
Anti-PL7 (%)	10 (5.2)
Anti-PL12 (%)	9 (4.8)
Anti-NXP2 (%)	6 (3.1)
Anti-SAE (%)	5 (2.6)
Anti-Ku (%)	5 (2.6)

Table 3 Therapies distribution in our cohort

Hydroxychloroquine (%)	105 (55)
Immunoglobulins (%)	96 (50.3)
Methotrexate (%)	95 (49.7)
Cyclophosphamide (%)	65 (34)
Mycophenolate mofetil (%)	60 (31.4)
Cyclosporine (%)	50 (26.2)
Rituximab (%)	35 (18.3)
Azathioprine (%)	25 (13.1)

P values < 0.05 were considered significant. The analysis was developed by using IBM SPSS statistics.

Results

The clinical charts of 191 patients were examined: 125 (65.4%) were female, the mean age was 66.6 ± 13.5 years and the mean disease duration was 9.8 ± 7.4 years. Epidemiological and clinical characteristics of the patients are reported in Table 1, while the distribution of their diagnosis is reported in Fig. 1.

The serological characteristics and therapies taken by patients are reported in Tables 2 and 3.

Ninety-nine patients (51.8%) had a TD; only 19 of them (9,9%) had developed TD after the diagnosis of IIM. Fifty-five patients (28.8%) had hypothyroidism, 3 (1.6%) hyperthyroidism and 41 (21.5%) had a normal thyroid function. Fifty-three (27.7%) had MNG, 42/191 (22%) had HT and 3/191 (1.6%) GD. One patient (0.5%) had a TPC that had been diagnosed several years before the diagnosis of IIM.

In total, 53 (27.7%) patients had proliferative disorders, 45 (23,6%) had metabolic TD and 1 (0.5%) patient had a neoplastic TD.

Those with hypothyroidism were taking a replacement therapy with levothyroxine. Four patients (2.1%)

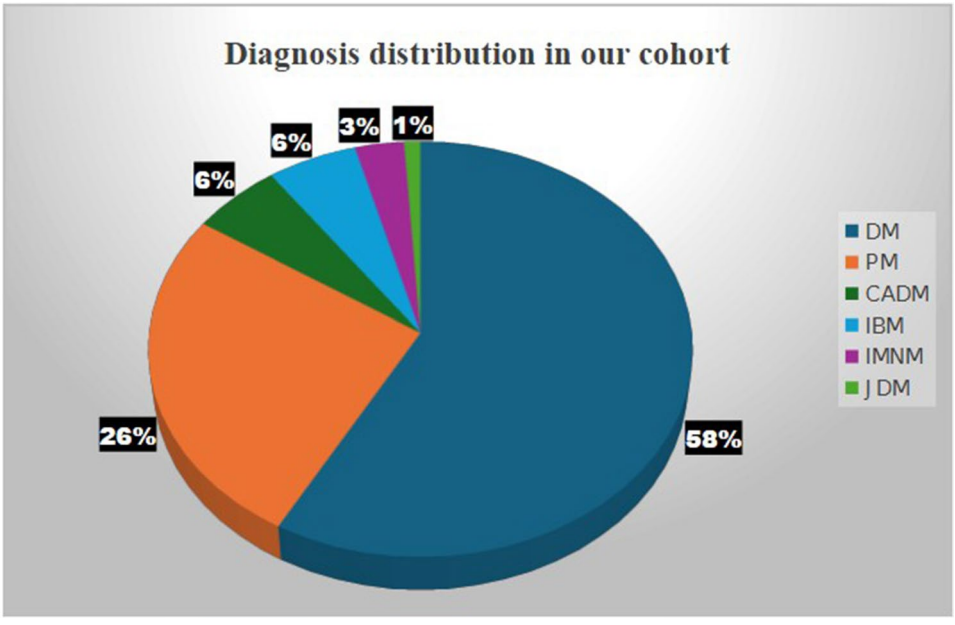

Fig. 1 Diagnosis distribution in our cohort. CADM: clinically amyopathic dermatomyositis; DM: dermatomyositis; IBM: inclusion body myositis; IMNM: immune-mediated necrotizing myopathy; JDM: juvenile dermatomyositis; PM: polymyositis

underwent total thyroidectomy (one patient with TPC and the other three with MNG).

No other concomitant autoimmune diseases were found in the cohort.

TDs occurrence was significantly associated with female sex ($p < 0.001$), older age (68.5 ± 11.3 vs. 64.5 ± 15.2, $p = 0.042$) and with a longer disease duration (10.8 ± 7.3 vs. 8.5 ± 7.2, $p = 0.03$).

No correlation was found between TDs and different subtypes of IIMs, nor with the different myositis-specific autoantibodies (MSA) and myositis-associated autoantibodies (MAA) (Table 4).

Among organ involvement the presence of a TD was significantly related with oesophageal involvement (67.7% vs. 51.1% $p = 0.014$); among the different comorbidities, TDs correlates with OP ($p < 0.001$) and FFx ($p = 0.014$) development.

We also observed that those who have TDs tended to accumulate a greater number of comorbidities than those without (6.6 ± 2.1 vs. 2.7 ± 2, $p < 0.001$). Moreover, patients with a TD showed significantly higher values of cumulative dose of glucocorticoids (GC) (13.7 ± 10.7 vs. 8.5 ± 8.8, $p = 0.032$).

Differentiating for each subtype of TD, HT occurrence was associated with female sex ($p < 0.001$) and OP development ($p = 0.002$), while MNG occurrence was associated with female sex ($p = 0.008$), older age ($p = 0.008$), arterial hypertension ($p = 0.013$) and OP development ($p = 0.005$). These correlations remained significant even in multivariate analysis.

The association between TDs and OP was independent from the total amount of GC ($p = 0.002$).

Table 4 MSA, MAA and IIM distribution based on the type of TD

Variable	HT	MNG	GD	TPC	*P*
Anti-Mi2	6	6	0	0	ns
Anti-TIF1 γ	2	7	0	1	ns
Anti-MDA5	4	2	0	0	ns
Anti-NXP2	2	2	0	0	ns
Anti-SAE	2	4	1	0	ns
Anti-Ku	1	4	0	0	ns
Anti-PMScl	8	12	0	0	ns
Anti-Jo1	4	6	0	0	ns
Anti-SRP	2	4	0	0	ns
Anti-PL7	4	6	1	0	ns
Anti-PL12	2	7	0	0	ns
Anti-Ro52	15	20	1	0	ns
DM	23	32	0	0	ns
PM	13	15	2	0	ns
CADM	3	3	0	1	ns
JDM	0	0	0	0	ns
IMNM	2	1	0	0	ns
IBM	2	2	0	0	ns

Finally, taking into account patients' QoL, the presence of a TD was associated with lower values of the SF-36 bodily pain domain (55.9 ± 27.1 vs. 71.6 ± 27.8, $p = 0.006$).

The characteristics of patients with and without TDs are summarized in Table 5.

Results of univariate and multivariate analysis are summarized in Table 6.

Discussion

Thyroid plays a pivotal role in myogenesis, muscle regeneration and contractile functionality, by modifying myocyte transcripts and by modulating bioenergetic metabolism [19].

The levels of T3, which represents the bioactive thyroid hormone, have been found to be associated with the functioning of the sarcoplasmic reticulum calcium ATPase (SERCA) pump, within myocytes [20]. Indeed, T3 promotes the pump activity, up-regulating calcium fluxes, thus influencing the energy turnover during the cycle of fibres contraction and relaxation [21].

The rates of the occurrence of every TD we analysed in this study was higher than those reported for GP, more significantly for MNG, HT and TPC.

The coexistence of TDs, particularly HT, in IIMs was initially described in some case reports [22, 23].

A common etiopathogenesis may be suitable as an explanation for coexistent IIMs and TDs. Proposed mechanisms might include shared environmental factors such as virus, drugs or chemicals [24], a cross reactivity of anti-thyroid autoantibodies or autoreactive T cells with other tissues and organs could lead to an overlap of the diseases [25], a genetic link between anti-thyroid autoimmunity and the susceptibility to autoimmune disease could play a crucial role [26].

Although the association between IIMs and hypo- and hyper-thyroidism was already reported [7], no data are actually available at our knowledge about the distribution of the different kinds of thyroid dysfunctions in this subgroup of patients.

It is already known that TDs tend to be more frequent in CTDs. Mosca et al. confirmed a higher prevalence of autoimmune TDs (34% of patients) in systemic autoimmune rheumatic diseases, particularly in SLE [27], while Antonelli and colleagues observed a higher prevalence of TPC in SLE patients than in the GP [28]. Moreover, Danieli et al. found that TSH levels were significantly higher in undifferentiated CTD patients than in healthy subjects [29].

In a meta-analysis, 35,708 patients with RA showed an increased risk of developing TDs, in particular hypothyroidism, with an odds ratio (OR) of 2.25 (95%CI 1.78–2.84) [30].

In Primary Sjögren's Syndrome Girón-Pïllado et al. reported a prevalence of TDs in 40% of patients [31].

Table 5 Characteristics of patients with and without TD

Variable	TD (*n* = 99)	NO TD (*n* = 92)	*P*
Female/Male	81/18	44/48	< 0.001
Mean age (year ± SD)	68.5 ± 11.3	64.5 ± 15.2	0.042
Mean age at onset (year ± SD)	57.6 ± 12.6	56.2 ± 17.1	ns
Mean age at diagnosis (year ± SD)	59.3 ± 18.1	56.1 ± 20.3	ns
Mean disease duration (year ± SD)	10.8 ± 7.3	8.5 ± 7.2	0.033
Skin involvement (%)	50 (50.5)	50 (54.3)	ns
Muscle involvement (%)	83 (83.8)	78 (84.8)	ns
Lung involvement (%)	44 (44.4)	34 (37)	ns
Raynaud phenomenon (%)	48 (48.5)	35 (38)	ns
Heart involvement (%)	7 (7.1)	5 (5.4)	ns
Oesophagus involvement (%)	67 (67.7)	47 (51.1)	0.014
Articular involvement (%)	24(24.2)	23 (25)	ns
Osteoporosis (%)	51 (51.5)	19 (20.7)	< 0.001
Fragility fractures (%)	21 (21.2)	8 (8.7)	0.014
Diabetes (%)	13 (13.1)	15 (16.3)	ns
Obesity (%)	12 (12.1)	12 (13)	ns
Cataract (%)	15 (15.1)	7 (7.7)	ns
Chronic renal failure (%)	11 (11.1)	7 (7.7)	ns
Dysthymia (%)	19 (19.2)	8 (8.7)	ns
Fibromyalgia (%)	15 (15.1)	4 (4.3)	ns
Neoplasms (%)	10 (10.1)	11 (12)	ns
Hyperuricemia (%)	36 (36.4)	31 (33.7)	ns
Gout (%)	1 (1)	6 (6.5)	ns
Myocardial infarction (%)	8 (8.1)	3 (3.3)	ns
Hypertension (%)	55 (55.6)	44 (47.9)	ns
Stroke (%)	3 (3)	0 (0)	ns
Atrial fibrillation (%)	6 (6.1)	4 (4.3)	ns
Dyslipidaemia (%)	47 (47.5)	37 (40.2)	ns
Mean HAQ (± SD)	0.7 ± 0.8	0.4 ± 0.6	ns
Mean Anxiety value (± SD)	5.6 ± 4.6	5.2 ± 4.4	ns
Mean Depression value (± SD)	5.8 ± 5.3	5.2 ± 4.9	ns
Mean FACIT-F (± SD)	3.9 ± 3.1	3.2 ± 3	ns
Mean SF36-PF (± SD)	53.8 ± 34.6	59.7 ± 30.4	ns
Mean SF36-RP (± SD)	37.7 ± 44.5	48.6 ± 45	ns
Mean SF36-RE (± SD)	49.3 ± 45.9	51.3 ± 44.8	ns
Mean SF36-VT (± SD)	51.1 ± 24.9	56.9 ± 23.3	ns
Mean SF36-MH (± SD)	65.3 ± 25.8	67.2 ± 20.7	ns
Mean SF36-SF (± SD)	67.6 ± 27.8	70.8 ± 20.9	ns
Mean SF36-BP (± SD)	55.9 ± 27.1	71.6 ± 27.8	0.006
Mean SF36-GH (± SD)	44.3 ± 22.3	48.6 ± 22.4	ns

Finally, recent data from a review on Systemic Sclerosis reported the incidence of hypothyroidism ranging from 4% to 33.3% of cases [32].

Exploring the role of TDs in influencing CTDs' clinical manifestations or comorbidities, Carli et al. observed the correlation with a higher risk of OP and FFx in SLE patients [10], while Cherim and colleagues observed a relationship with digital ulcers, calcinosis, pulmonary arterial hypertension, scleroderma renal crisis and Raynaud's phenomenon development in patients with SSc [32].

Table 6 Univariate and multivariate analysis

Variable	Univariate Analysis	Multivariate Analysis
Female sex	TDs: $p < 0.001$	-
Older age (years)	TDs: $p = 0.042$	TDs: $p = 0.03$
Longer disease duration (years)	TDs: $p = 0.03$	–
Oesophageal involvement	TDs: $p = 0.014$	–
Osteoporosis	TDs: $p < 0.001$	TDs: $p = 0.002$
Fragility fractures	TDs: $p = 0.014$	-
Greater number of comorbidities	TDs: $p < 0.001$	–
Greater cumulative glucocorticoid dose (GC)	TDs: $p = 0.032$	–
Lower values of SF-36 Bodily Pain domains	TDs: $p = 0.006$	–

Considering the demographic characteristics of patients, our results confirmed a higher risk of TDs in women.

When evaluating IIM's clinical involvement, we observed a statistically significant association between oesophageal involvement and TDs. This observation is newsworthy, since several works in the literature have shown an association between oesophageal achalasia and TDs and it has been reported that compared to GP, patients with achalasia were 8.5 times as likely to have hypothyroidism. Furthermore, although the aetiology of achalasia is unknown, autoimmunity could be implicated, as already supported by several studies [33, 34] Finally, hypothyroidism could affect oesophagus motility via both shortened duration and reduced percentage of relaxation, even in patients without any gastrointestinal symptoms [35].

Interestingly, in our IIM patients the presence of TDs appeared associated also with a higher risk of developing comorbidities. In particular, similarly to what already observed in SLE, TDs are associated with an increased prevalence of both OP and FFx occurrence [10].

Both IIMs and TDs are known as risk factors for OP [11, 36]. The mechanisms on which this relationship is based are likely multifactorial, including systemic inflammation, a treatment with GC, a reduced mobility and an impaired calcium/vitamin D homeostasis [37, 38].

Although OP risk was associated with TDs independently from the total amount of GC, the association between TDs and higher steroids doses could partially explain their tendence to accumulate more comorbidities, that often could derive from GC toxicity (for example hypertension or dyslipidaemia). No data are available at the moment about this association; therefore, additional analysis in larger cohorts is advisable, to clarify this result.

Considering the different kinds of TDs, our data confirmed the association of both HT and MNG with female

sex and OP occurrence; moreover, they confirmed also the relationship between MNG and hypertension [38].

The prevalence of TPC seems significantly higher into the analyzed IIM cohort than in GP. However, taking into account that only one patient had developed a TPC, more data from larger cohorts of IIM patients are needed to confirm this data.

TDs were shown to have an impact also on QoL, as the occurrence of a thyroid pathology was associated with worse values of the physical pain domain of SF36. This finding agrees with some previous papers on people with TDs, showing that the optimization of thyroid function could determine a global improvement of patients [39–41].

Although we tried to analyse a relatively unexplored subject in patients with IIMs, our study surely has some limitations. At first, it is retrospective; moreover, we studied a monocentric cohort; finally, only a limited number of patients had a diagnosis of GD and TPC.

Conclusion

TDs tend to associate with IIMs, reasonably in relation with several shared pathogenetic aspects and with possible consequences both on patients' clinical picture and QoL.

Our results partially fill the lack of knowledge about the relationship between TDs and IIMs; furthermore, they suggest the opportunity to adhere to shared endocrinological guidelines of thyroid function assessment, both for blood exams and targeted ultrasound, during the regular assessment of IIM patients.

Indeed, a thyroid dysfunction could not only aggravate muscle impairment, but also widely influence the clinical spectrum of IIMs patients, from disease activity, to damage, passing by comorbidities and QoL, thus worsening their already complex disease burden (see Table 7).

Table 7 Influence of TDs on IIMs

Influence of TDs on IIMs patients	
Activity	Esophageal involvement
Damage	OP
Comorbidities	Hypertension, accumulation of comorbidities
Quality of Life	Increased pain perception

Abbreviations

IIMs	Idiopathic Inflammatory Myopathies
TDs	Thyroid Diseases
HT	Hashimoto Thyroiditis
MNG	Multinodular Goitre
GD	Grave's Disease
TPC	Thyroid Papillary Cancer
QoL	Quality of Life
DM	Dermatomyositis
PM	Polymyositis
CADM	Clinically Amyopathic Dermatomyositis
IBM	Inclusion Body Myositis
IMNM	Immune-Mediated Necrotizing Myopathy
JDM	Juvenile Dermatomyositis
SF-36	Short Form 36
HAQ	Health Assessment Questionnaire
HADS	Hospital Anxiety and Depression Scale
FACIT-F	Functional Assessment of Chronic Illness Therapy-Fatigue
MSA	Myositis-Specific Autoantibodies
MAA	Myositis-Associated Autoantibodies
OP	Osteoporosis
FFx	Fragility Fractures
GC	Glucocorticoids

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Author contributions

LC: concept, method, writing, review&editing, formal analysis, project administ, data curation, supervision, visualization MD: writing original draft, formal analysis, data curation, investigation. CC, FF, EL: investigating, data curation. SB: formal analysis, visualization AT: visualization MM: review&editing, supervision, project administration.

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Data availability

data and materials are available from the corresponding author upon request.

Declarations

Ethics approval and consent to participate

the study was approved by the local Ethics Committee [(Comitato Etico Regionale per la Sperimentazione Clinica della Regione Toscana – AREA VASTA NORD OVEST (Regional Ethics Committee for Clinical Research of the Tuscany Region – NORTH-WEST AREA); Institute: Unità Sanitaria Locale Toscana Nord Ovest (Local Health Authority of North-West Tuscany)], and the committee's reference number is 20070.

Patient consent for publication

all patients provided consent for data publication.

Informed consent

All participants signed an informed consent prior to inclusion in the study.

Competing interests

The authors declare no competing interests.

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