IL-6	8,1	39,7	<0.0001
IL-8	2,4	3,4	0.169
IL-13	5,4	7,82	0.005
G-CSF	20,9	278	<0.0001
HGF	73,0	109,7	<0.001
MCP-1	53,6	64,8	0.01
IL-1 α	1,2	9,1	<0.001
FGF-2	2,9	25,5	<0.001
VEGF	32,3	406,6	<0.001
PDGF- β	8,2	329,7	<0.001

Table 2. Values of growth factors in platelet rich plasma (PRP), *n* patients= 23, controls= 16

Factor		Mean (pg/ mL)*	p
TFG- β 1	Patients	220271,8	0,327
Controls	261498,1		
IL-1 β	Patients	111,3	0,492
Controls	125,3		
IL-6	Patients	41,4	0,575
Controls	37,2		
IL-8	Patients	2,4	0,159
Controls	2,4		
IL-13	Patients	8,1	0,574
Controls	7,4		
G-CSF	Patients	282,0	0,820
Controls	272,2		
HGF	Patients	105,5	0,598
Controls	115,9		
MCP-1	Patients	66,9	0,841
Controls	61,8		
IL-1 α	Patients	8,4	0,517
Controls	10,1		
FGF-2	Patients	29,4	0,257
Controls	19,9		
VEGF	Patients	248,9	<0.0001***
Controls	633,3		
PDGF- β	Patients	270,6	0,12
Controls	414,7		

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AB0204 THE CLINICAL FEATURES OF ANTI-TIF1GAMMA ANTIBODY POSITIVE DERMATOMYOSITIS

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Background: Anti-TIF1gamma antibody positive dermatomyositis (TIF1 γ -DM) has greater risk of complication with cancer.

Objectives: We aimed to analyze the clinical features of TIF1 γ -DM and find the difference between cancer-associated TIF1 γ -DM (CA-TIF1 γ -DM) and non-cancer-associated TIF1 γ -DM (nCA-TIF1 γ -DM).

Methods: We investigated the clinical data of idiopathic inflammatory myositis (IIM) patients received remission induction therapy in out hospital between January 2006 and December 2018.

Results: We treated 148 patients with IIM in this period, 9 cases of them were TIF1 γ -DM. Patients with CA-TIF1 γ -DM and nCA-TIF1 γ -DM were 4 and 5 cases, respectively. Average age at onset of 9 patients with TIF1 γ -DM was 63 years old. CA-TIF1 γ -DM was significantly older than nCA-TIF1 γ -DM (average:78.0 vs 57.4). All of 9 TIF1 γ -DM had distinctive skin rash and muscle weakness. Seven cases of them suffered from dysphagia. Interstitial lung disease was not seen in all patients with

TIF1 γ -DM. Serum creatine kinase (CK) levels of CA-TIF1 γ -DM was significantly higher than those of nCA-TIF1 γ -DM (average: 3405.5IU/L vs 719.8IU/L) (Fig 1.). Among the TIF1 γ -DM patients, we administered glucocorticoid (GCs) for 8 cases, immunosuppressants for 4 cases, and intravenous immunoglobulins (IVIg) for 7 cases. All 4 cases of CA-TIF1 γ -DM were treated with GC and IVIg. All TIF1 γ -DM patients, especially 4 CA-TIF1 γ -DM patients, seemed to have good response to treatment in serum CK levels. Although serum CK levels of these 4 cases were normalized within a month (Fig1.), their muscle weakness, skin rash and dysphagia which was associated with quality of life and the prognosis were poorly improved.

Figure 1: serum CK levels for 3months after treatment; Compared with cancer-associated TIF1 γ -DM (CA-TIF1 γ -DM) and non-cancer-associated TIF1 γ -DM (nCA-TIF1 γ -DM)

Figure 1

Conclusion: In our investigation, the age of onset and serum CK levels were significantly different between CA-TIF1 γ -DM and nCA-TIF1 γ -DM. We report our investigation of the phenotype and treatment of anti TIF1-gamma antibody positive dermatomyositis. TIF1 γ -DM with high serum CK levels, we should perform appropriate tests to check for cancer throughout our treatment, and the improvement of serum CK levels is not equal to the success of our treatment.

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AB0205 TREATMENT OF INFLAMMATORY MYOSITIS IN CLINICAL PRACTICE: ANALYSIS OF THE REGISTRY OF INFLAMMATORY MYOSITIS FROM THE MADRID COMMUNITY (REMICAM)

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Background: Although glucocorticoids (GC) remain the cornerstone in the treatment of inflammatory myositis (IM), the management of these processes is not yet standardized. Experts recommend early use of immunosuppressants (IS), in order to prevent GC side effects. It is not known what is the actual management in clinical practice.

Objectives: To describe the management of IM in REMICAM registry, and to identify differences according to disease subtype (polymyositis (PM), or dermatomyositis (DM)), and to the year of diagnosis (before or after 2000).

Methods: All patients from REMICAM were included (1). A descriptive analysis of the different therapies (GC, IS, biologics, IV immunoglobulins

(IVIG), and hydroxychloroquine HCL) was performed, attending to possible differences between IM subtype and the year of diagnosis. T student and Chi square tests were used for quantitative and qualitative variables, respectively.

Results: From 479 patients (74% females, 52% PM, 44±22 y at diagnosis, 10±8 y follow up), 473 (99%) received oral GC, 50/344 (15%) pulses GC, 78 (16%) HCL, 355 (74%) IS (methotrexate MTX 228, leflunomide 10, azathioprine AZA190, cyclophosphamide 44, mycophenolate MP 38, cyclosporine 32), 55 (12%) biologics (rituximab 45, abatacept 2, aTNF 16) and 79 (17%) IVIG. Differences according to IM subtype (1), and to the year of diagnosis (2), are shown in Tables.

Table 1

	PM (n=251; 52%)	DM (n=228; 48%)	p
mean ± SD			
Time (T) initial symptoms- diagnosis (dx) (m)	19±43	11±28	0.04
T initial - any treatment (tt) (m)	15±42	7±15	0.009
T initial - GC (m)	16±42	8±18	0.01
n; %			
MTX	104/251 (41%)	124/228 (54%)	0.006
AZA	114/251 (45%)	76/227 (33%)	0.009
Mycophenolate	30/244 (12%)	8/224 (4%)	0.001
HCL	27/251 (11%)	51/228 (22%)	0.001
GC withdrawal due to improvement	52/245 (21%)	100/212 (47%)	<0.0001
Severe infections	75/241 (31%)	38/217 (17%)	0.001

Table 2

	Diagnosis <2000 (n=208, 43%)	Diagnosis >2000 (n=271, 57%)	p
mean ± SD			
T initial - dx (m)	22±48	11±26	0.006
T initial - any tt (m)	16±42	8±22	0.02
T initial - GC (m)	17±43	9±24	0.02
T initial - IS (m)	53±82	14±28	<0.0001
T dx - IS (m)	39±75	4±19	<0.0001
N° tt	2.3±1.6	2.8±1.3	<0.0001
N° IS	0.9±1	1.3±0.8	<0.0001
n; %			
Any IS	121/208 (42%)	234/271 (86%)	<0.0001
MTX	69/208 (33%)	159/271 (59%)	<0.0001
MP	9/205 (4%)	29/263 (11%)	0.01
Any biologic	12/208 (6%)	43/271 (16%)	<0.0001
RTX	8/208 (4%)	37/271 (14%)	<0.0001
IVIG	21/204 (10%)	58/271 (21%)	0.001
GC withdrawal due to improvement	75/189 (40%)	77/191 (29%)	0.02
All cause mortality	69/162 (43%)	45/247 (18%)	<0.0001
Severe infections	61/192 (32%)	52/266 (19%)	0.003

Conclusion: The therapeutic management of IM has changed over the last few years, with a clear increase and earlier use of IS in patients diagnosed after 2000. In spite of a more aggressive approach in recent years, severe infections are less frequent, perhaps as a consequence of lower GC doses. The management of PM and DM is similar.

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AB0206

IS DISEASE SEVERITY A RISK FACTOR FOR CARDIOVASCULAR MORTALITY IN INFLAMMATORY MYOSITIS? ANALYSIS OF THE REGISTRY OF INFLAMMATORY MYOSITIS FROM THE MADRID COMMUNITY (REMICA)

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Background: Inflammatory myositis (IM) are heterogeneous autoimmune diseases, characterized by muscular inflammation, which can associate systemic manifestations. In other inflammatory diseases, as rheumatoid arthritis, inflammation is a cardiovascular risk factor (CVRF), with clear influence on cardiovascular events and mortality (CVE, CVM).

Objectives: To analyze the influence of IM severity in the development of CVE and CVM in the REMICA registry.

Methods: All patients from REMICA were included(1). REMICA is a retrospective multicentric registry of IM patients (n=479), performed in Madrid between 2012 and 2014, containing demographic, clinical and outcome data (1). The presence of extra muscular features (cardiac, pulmonary, cutaneous and systemic involvement), number of therapies, immunosuppressants (IS) need, and glucocorticoids (GC) withdrawal due to IM remission defined IM severity. Bi and multivariate logistic regression analysis were used to determine the association between these factors and CVE. Survival analysis and regression proportional hazard bi and multivariate Cox models were used to determine the effect of these factors on CVM. All factors with p< 0,2 in bivariate analysis were included in multivariate models. The effect was controlled for age, sex and CVRF.

Results: From 479 patients (74% females, 52% polymyositis, 44±22 years at diagnosis, 10±8 years follow up), 104/467 (22%) presented CVE and 24/409 (6%) died due to CVE. CVE were associated to age, male sex, CVRF number, diagnosis before year 2000, presence of arrhythmia or Raynaud (Table 1). Arrhythmia was the only independent risk factor for CVM. GC withdrawal due to IM remission, and a smaller number of therapies, were protective factors for CVM (Table 2).

Conclusion: In the REMICA registry, extra muscular manifestations such as arrhythmia, were associated to CVE and CVM, as independent risk factors. A smaller number of treatment or GC withdrawal due to remission were protective factors for CVM. Our results support the hypothesis that more severe IM, with extra muscular involvement and persistent inflammation, could favor atherosclerosis and CVE development.

Table 1. Characteristics associated with cardiovascular event (ECV) development

	Bivariate		Multivariate	
	OR (IC 95%)	P	OR IC 95%)	p
Sex	1.7 (0.9-2.9)	0.06	3.2 (1.4-7.4)	0.007
Age at diagnosis	1.04 (1.03-1.05)	<0.0001	1.04 (1.02-1.06)	<0.0001
CVRF number	2.2 (1.7-2.7)	<0.0001	1.9 (1.4-2.5)	<0.0001
Raynaud	2.01 (1.3-3.2)	0.003	2.3 (1.2-4.6)	0.015
Arrhythmia	9.7 (5.2-18.1)	<0.0001	5.7 (2.4-13.4)	<0.0001
Pericarditis	3.9 (1.8-8.3)	<0.0001		0.06
Diagnosis after year 2000	0.7 (0.5-1.08)	0.1	0.4 (0.2-0.7)	0.004

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[1] Nuño L, Rheumatol Clin 2017; 13:331-337

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