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Subclinical Coronary Artery Disease in Patients with Idiopathic Inflammatory Myopathy: An Evaluation by CT Coronary Angiography

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ABSTRACT

Idiopathic inflammatory myopathy (IIM) poses elevated risk of cardiovascular event and mortality, similar to other autoimmune rheumatic diseases. We conducted a cross-sectional study to examine the prevalence and risk factor of subclinical coronary artery disease (CAD) in patients with IIM, using CT coronary angiogram (CTCA). The prevalence of obstructive CAD and CAD in IIM (13.3% and 66.7%, respectively) were significantly higher than age and sex-matched controls (0% and 30%, respectively, both $p < 0.05$). Diabetes mellitus and calcium calcification score > 100 units were found to be the independent predictors of obstructive CAD. Screening of high-risk patients with aggressive treatment of cardiovascular risk factors should be considered in managing IIM patients.

Keywords: Myositis; Cardiovascular Disease.

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INTRODUCTION

Idiopathic inflammatory myopathy (IIM) patients are well-known to have high mortality and morbidity [1–4]. The cardiovascular mortality in IIM among all causes of mortality ranged from 14% to 55% [5–7], with coronary artery disease (CAD) as the major cause. A study in Denmark showed that up to 20% IIM patients suffered from significant coronary calcification by computed tomography (CT), fivefold higher risk compared to healthy controls [8].

There was a paucity of data of CAD in asymptomatic patients with IIM. This study aimed to investigate the prevalence of subclinical CAD in IIM patients, compared with age-/sex-matched controls. The secondary outcome was to identify risk factors of obstructive CAD in IIM patients.

PATIENTS AND METHODS

Study design and participants

This was a cross-sectional study enrolling IIM patients consecutively followed up in Kwong Wah Hospital from 2020 to 2021. IIM patients fulfilling Bohan and Peter Criteria [9], with age >18 and without preexisting cardiovascular disease history, were recruited. IIM patients with history of active malignancy, disease duration less than 1 year, overlap syndrome with

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other rheumatological diseases, renal impairment with estimated glomerular filtration rate (eGFR) <50 mL/min, CAD symptoms, ischemic changes on electrocardiography (ECG), or impaired left ventricular ejection fraction <40% on echocardiogram were excluded. Thirty-two patients fulfilling these criteria were recruited, with two patients dropped out of the study due to personal reason prior to CT coronary angiogram (CTCA).

Anonymous clinical data and CTCA reports of 30 age-matched and sex-matched individuals were randomly retrieved from the data base of the same image center from 2020 to 2021 retrospectively. To minimize selection bias, patients with a known history of cardiovascular disease, and typical CAD symptoms, including chest pain and abnormal ECG as the indications of CTCA, were excluded. The typical indications of CTCA in these controls were cardiovascular risk assessment or body check before COVID vaccination.

Written consents were obtained. The objectives, procedures, and complications were informed and documented in the consent form. The study was approved by the local Ethics Committee of the Hospital Authority. The study was conducted in full compliance with the Helsinki Declaration. CTCA results were subsequently disclosed to the patients with suggestion of treatment plan, including risk factor control, medical therapy, or referral to cardiac team for further management whenever deemed necessary.

Clinical assessment

At the recruitment of the IIM patients, demographic data, including age, sex, body mass index, smoking status, and quantities in packs-year, if any, medical history, drug history, and family history of immature cardiovascular disease (before 55 and 65 for the first degree of male and female, respectively), were obtained during face-to-face interview or from the Hospital Authority Clinical Management System (CMS). The system has been extensively used in epidemiological studies involving IIM patients [10]. Biochemical markers including total cholesterol (TC), low-density lipoprotein cholesterol (LDL), high-density lipoprotein cholesterol (HDL), triglyceride (TG), fasting glucose (FG), HbA1c, and renal function within 3 months prior to CTCA were retrieved or repeated if necessary. Diabetes mellitus was defined by ever HbA1c ≥ 6.5%, FG ≥ 7 mmol/L, or use of hypoglycemia agent. Hypertension was defined by systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥ 90 mmHg, or use of antihypertensive agents. Hyperlipidemia was defined by TC ≥ 5.0 mmol/L, TG ≥ 1.7 mmol/L, LDL ≥ 3.0 mmol/L, or use of lipid-lowering therapy.

Myositis disease measures

IIM patients were classified, according to the 2017 American College of Rheumatology (ACR)/European League Against Rheumatism (EULAR) classification criteria [11], into amyopathic dermatomyositis, dermatomyositis, and polymyositis.

Immune-mediated necrotizing myopathy (IMNM) was grouped under the polymyositis subgroup for analysis. Health Assessment Questionnaire (HAQ) and Manual Muscle Test (MMT8) were performed, covering patient-reported subjective functional scores and physician objective muscle strength assessments, respectively. Biochemical markers, including maximum and current level of muscle enzyme creatine kinase (CK) and lactate dehydrogenase (LDH), myositis autoantibodies profile (MSA), were retrieved from CMS for analysis.

Medication history included the maximum daily dose, duration, cumulative and current dose of glucocorticoid, and any history of second-line immunosuppressive agents used were recorded.

Coronary atherosclerosis assessment

CTCA was performed in a single center with images analyzed by a single radiologist to minimize interobserver error. Calcium calcification was quantified by Agatston method [12]. The most stenotic plaque among the three coronary arteries was recorded. Obstructive CAD was defined by level of stenosis more than 50%.

Statistical analysis

Statistical analyses were performed by version 24.0 of the Statistics Package for the Social Sciences (SPSS). Descriptive statistics were presented as a count (percentage), mean ± standard deviation or median with a range if appropriate. Comparative statistics were performed by Chi-Square test or Fisher Exact test, and independent *t* test or Mann–Whitney *U* test according to the data distribution. Univariate analysis and multivariate analysis via backward logistic regression were performed to analyze the odds ratio of variables in the secondary outcome assessment. Results were considered statistically significant if $p < 0.05$.

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Table 1. Demographics of IIM patients and control

		IIM (n = 30)	Control (n = 30)	P-value
Demographics	Age	56.6 ± 10.8	56.2 ± 11.1	0.906
	Female n (%)	17 (56.7)	17 (56.7)	1
Comorbidities	Diabetes mellitus	5 (16.7)	3 (10)	0.448
	Hypertension	7 (23.3)	6 (20)	0.754
	Hyperlipidemia	17 (56.7)	4 (13.3)	<0.001*

* Significant value with $p < 0.05$.

Table 2. Characteristic of IIM patients

		IIM (n = 30)
Body mass index		23.3 ± 2.8
Active smoker n (%)		5 (16.7)
Subtype	Polymyositis n (%)*	11 (36.7)
	Dermatomyositis n (%)	9 (30)
	Amyopathic dermatomyositis n (%)	10 (33.3)
	IMNM n (%)	4 (13.3)
	Duration of disease (Yr)	6.0 (3.0–14.0)
Biochemical markers	TC (mmol/L)	5.1 ± 0.9
	LDL (mmol/L)	2.8 ± 0.7
	HDL (mmol/L)	1.6 ± 0.4
	TG (mmol/L)	1.3 (1.0–1.7)
	FG (mmol/L)	5.2 (4.7–5.7)
	HbA1c (%)	5.8 (5.5–6.1)
Medication	Duration of steroid (Yr)	3.0 (2.0–8.3)
	Current daily dose of steroid (mg)#	5.0 (0.8–6.4)
	Cumulative dose of steroid (mg)#^	14,260 (8,000–19,690)
	Number of second-line therapy used	2.0 (1.8–4.0)
Disease activity	HAQ	0.13 (0.0–0.53)
	MMT8	78.0 (75.5–80.0)
	Current CK	115 (97–228)
	Current LDH	216 (191–255)

IMNM: Immune-Mediated Necrotizing Myopathy; TC: Total Cholesterol; LDL: Low-Density Lipoprotein Cholesterol; HDL: High-Density Lipoprotein Cholesterol; TG: Total Triglyceride; FG: Fasting Glucose; HAQ: Health Assessment Questionnaire; MMT8: Manual Muscle Test 8; CK: Creatine Kinase; LDH: Lactate Dehydrogenase.

* Includes IMNM.

Prednisolone-equivalent dose.

^ Rounded off to the nearest 10.

RESULTS

A total of 30 IIM patients and 30 controls were recruited. The demographic data and underlying comorbidities in IIM and control groups are summarized in Table 1. The mean age of the IIM patients was 56.6 ± 10.8 with 56.7% of them being female. Many IIM patients had

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Table 3. CTCA outcome in IIM patients and control

	IIM (n = 30)	Control (n = 30)	P-value
CAC score	2.6 (0–77)	0 (0–0)	<0.001*
Presence of CAD n (%)	20 (66.7)	9 (30.0)	0.004*
Presence of obstructive CAD n (%)	4 (13.3)	0 (0)	0.038*

CAC: Coronary Artery Calcium; CAD: Coronary Artery Disease.
* Significant value with $p < 0.05$.

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hypertension, hyperlipidemia, or diabetes mellitus (56.7%, 23.3%, and 16.7%, respectively). More IIM patients had hyperlipidemia than the control group (56.7% vs. 13.3%, $p < 0.001$).

The clinical characteristics of IIM patients are shown in Table 2. The median duration of IIM disease and steroid therapy received were 6.0 years (3.0–14.0) and 3.0 years (2.0–8.3), respectively. Median of current daily and cumulative dose of prednisolone received were 5.0 mg (0.8–6.4) and 14,260 mg (8,000–19,690), respectively. A small proportion of the patients (16.7%) were active smokers. Median MMT8 and HAQ were 78.0 (75.5–80.0) and 0.13 (0.0–0.53), respectively.

Using CTCA, 20 IIM patients (66.7%) had CAD of various degrees. Obstructive CAD was found in four (13.3%) asymptomatic patients with IIM. The presence of both obstructive and all degree CAD was significantly higher in IIM cohort compared to control (0%, $p = 0.038$; 30%, $p = 0.004$, respectively). The median coronary artery calcium (CAC) score was also significantly higher

Table 4. Characteristics of IIM patients subdivided according to the degree of CAD

		IIM with obstructive CAD (n = 4)	IIM without obstructive CAD (n = 26)	P-value§	IIM with nonobstructive CAD (n = 16)	P-value¶
Demographic	Age	60.5 ± 9.6	56.0 ± 11.0	0.44	60.0 ± 11.1	0.94
	BMI	23.5 ± 3.0	23.3 ± 2.8	0.88	23.3 ± 3.2	0.92
	Female n (%)	3 (75)	14 (53.8)	0.43	9 (56.3)	0.49
	Active smoker n (%)	2 (50)	4 (15.4)	0.11	3 (18.8)	0.20
	Packs-yr (Ever smoker)	36.5 (30–43)	10 (8–30)	0.07	10 (7–22.5)	0.08
Comorbidities	Diabetes mellitus n (%)	3 (75)	2 (7.7)	0.001*	2 (12.5)	0.01*
	Hypertension n (%)	4 (100)	3 (11.5)	<0.001*	3 (18.8)	0.002*
	Hyperlipidemia n (%)	4 (100)	13 (50)	0.06	8 (50)	0.07
Blood pressure	Systolic blood pressure (mmHg)	132 ± 12	125 ± 12	0.25	125 ± 10	0.19
	Diastolic blood pressure (mmHg)	79 ± 16	75 ± 7	0.67	73 ± 7	0.55
Biochemical markers	TC (mmol/L)	5.4 ± 1.6	5.1 ± 0.8	0.51	5.1 ± 0.8	0.65
	LDL (mmol/L)	3.0 ± 0.9	2.8 ± 0.7	0.56	2.8 ± 0.6	0.59
	HDL (mmol/L)	1.6 ± 0.7	1.6 ± 0.4	0.95	1.6 ± 0.4	0.95
	TG (mmol/L)	1.7 ± 0.5	1.4 ± 0.5	0.29	1.4 ± 0.6	0.34
	FG (mmol/L)	6.6 (5.1–7.0)	5.2 (4.7–5.5)	0.1	5.3 (4.8–5.6)	0.14
	HbA1c %	7.4 ± 1.4	5.8 ± 0.4	0.11	5.8 ± 0.5	0.11
	Maximum CK (IU/L) ^	5,920 (1,740–21,730)	320 (130–3,380)	0.03*	280 (140–3,430)	0.03*
	Maximum LDH (IU/L) ^	800 (490–1,740)	400 (330–520)	0.04*	420 (330–510)	0.07

(*Continued*)

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Table 4. (*Continued*)

		IIM with obstructive CAD (*n* = 4)	IIM without obstructive CAD (*n* = 26)	*P*-value§	IIM with nonobstructive CAD (*n* = 16)	*P*-value¶
	Current CK (IU//L)	129 (89–2,790)	115 (97–228)	0.659	115 (85–169)	0.62
	Current LDH (IU/L)	219 (204–444)	216 (284–255)	0.425	209 (180–249)	0.34
Functional assessment	HAQ	0.26 (0.03–0.57)	0.07 (0–0.53)	0.61	0.13 (0–0.60)	0.73
	MMT8	74 (70–79)	79 (78–80)	0.19	78 (78–80)	0.20
Disease	Polymyositis *n* (%) ^	4 (100)	7 (26.9)	0.005*	5 (31.3)	0.01*
	Duration of disease (Yr)	10.3 ± 5.5	7.5 ± 6.3	0.42	8.3 ± 7.0	0.60
Medication	Duration of steroid (Yr)	10.0 (4.5–10.3)	3.0 (2.0–6.3)	0.12	3.5 (1.3–6.8)	0.18
	Maximum daily dose of steroid (mg)#	52.3 (41.3–60.0)	50.0 (28.8–50.0)	0.28	50.0 (36.3–50.0)	0.31
	Current daily dose of steroid (mg)#	5.0 (2.8–5.8)	5.0 (0.0–7.5)	0.95	4.0 (0.0–7.5)	0.67
	Cumulative dose of steroid (mg)^#	19,830 (14,300–24,770)	12,970 (7,490–19,180)	0.13	12,970 (5,320–18,970)	0.16
	Number of second-line therapy used	2.5 (1.3–3.8)	2.0 (1.8–4.3)	0.85	2.0 (1.3–3.5)	0.80
Assessment	Framingham score (%)	27.4 (18.1–30.0)	9.7 (3.3–11.7)	0.005*	11.2 (6.3–20.2)	0.02*
	ASCVD (%)	15.4 (13.9–25.7)	2.9 (1.5–6.3)	0.003*	6.0 (2.6–8.9)	0.01*
	Ejection fraction (%)	60.5 ± 11.4	65.7 ± 8.4	0.28	67.0 ± 8.8	0.25
	CAC score	243 (56–415)	NA	NA	61 (2–90)	0.14

TC: Total Cholesterol; LDL: Low-Density Lipoprotein Cholesterol; HDL: High-Density Lipoprotein Cholesterol; TG: Total Triglyceride; FG: Fasting Glucose; CK: Creatine Kinase; LDH: Lactate Dehydrogenase; HAQ: Health Assessment Questionnaire; MMT8: Manual Muscle Test 8; ASCVD: Atherosclerotic Cardiovascular Risk Score; CAC: Coronary Artery Calcium; Yr: Year.

^ Rounded off to the nearest 10.

Prednisolone-equivalent dose.

§ Represent the *p*-value comparing the subgroup of obstructive CAD with the subgroup without obstructive CAD.

¶ Represent the *p*-value comparing the subgroup of obstructive CAD with the subgroup of nonobstructive CAD subgroups.

* Significant value with $p < 0.05$.

^ Include IMNM.

Table 5. Logistic regression of IIM patients with obstructive CAD

	Univariate analysis		Multivariate analysis	
	Unadjusted OR (95% CI)	*P*-value	Adjusted OR (95% CI)	*P*-value
Diabetes mellitus	36 (2.46, 527.1)	0.009*	34.5 (2.35, 505.75)	0.01*
Active smoker	5.5 (0.59, 51.9)	0.134	NS	NS
FG	6.3 (1.25, 31.89)	0.026*	NA	NA
HbA1c	33.6 (1.20, 936.99)	0.039*	NA	NA
Duration of steroid	1.16 (0.95, 1.42)	0.141	1.03 (0.95, 1.80)	0.11
Maximum CK/100	1.01 (1.00, 1.02)	0.089	NS	NS
Maximum LDH/100	1.26 (0.99, 1.61)	0.057	NS	NS
CAC score ≥ 100	23 (1.78, 298.45)	0.016*	22 (1.69, 285.89)	0.018*

FG: Fasting Glucose; CK: Creatine Kinase; LDH: Lactate Dehydrogenase; CAC: Coronary Artery Calcium; NA: Not Applicable; NS: Nonsignificant.

Prednisolone-equivalent dose.

* Significant value with $p < 0.05$.

in the IIM group compared to the control group (2.6 vs. 0, $p < 0.001$). Table 3 summarizes the CTCA outcome in both IIM and control groups.

IIM patients with obstructive CAD had higher prevalence of comorbidities including diabetes mellitus, hypertension, and hyperlipidemia than IIM patients without obstructive CAD (diabetes: 75% vs. 7.7%, $p = 0.001$; hypertension 100% vs. 11.5%, $p < 0.001$; hyperlipidemia 100% vs. 50%, $p = 0.06$; Table 4). FG and HbA1c were also numerically higher in IIM with obstructive CAD subgroup [FG: 6.6 mmol/L (5.1–7.0) vs. 5.2 mmol/L (4.7–5.5), $p = 0.1$; HbA1c 7.4 ± 1.4% vs. 5.8% ± 0.4%, $p = 0.11$]. The maximum level of muscle enzyme (CK and LDH) in IIM with obstructive CAD subgroup were both significantly higher than IIM without obstructive CAD subgroup [CK: 5,920 IU/L (1,740–21,730) vs. 320 IU/L (130–3,380), $p = 0.03$; LDH 800 IU/L (490–1,740) vs. 400 IU/L (330–520), $p = 0.04$]. Regarding subtype of IIM, polymyositis was more commonly found in IIM with obstructive CAD ($n = 4$, 100%) than IIM without obstructive CAD (26.9%), with a p-value of 0.005. With regards to immunosuppressants received, IIM with obstructive CAD received numerically higher cumulative prednisolone [19,830 mg (14,300–24,770) vs. 12,970 mg (7,490–19,180), $p = 0.13$], longer duration of steroid [median 10.0 years (4.5–10.3) vs. 3.0 years (2.0–6.3), $p = 0.12$]. Second-line immunosuppressants use was similar.

Table 5 shows the univariate and multivariate analysis results of risk factors of obstructive CAD in IIM. In the multivariate analysis, only the presence of diabetes mellitus (OR 34.5, 95% CI 2.35, 505.75, $p = 0.01$) and CAC score ≥ 100 units (OR 22, 95% CI 1.69, 285.89, $p = 0.018$) were independently associated with obstructive CAD.

DISCUSSION

This was the first study to evaluate the prevalence of subclinical CAD in a cohort of IIM patients using CTCA. The higher prevalence of subclinical CAD in IIM patients compared with control is consistent with other immune-mediated disease such as inflammatory arthritis [13] or SLE [14]. This underscores the possibility of systemic inflammation being the common pathogenic pathway of accelerated atherosclerosis. The prevalence of obstructive CAD and CAC scores was also significantly higher in the IIM group. The cardiovascular disease burden of IIM should not be overlooked.

While the association of CAD in IIM was commonly found in studies [4,15] the relationship of specific phenotypic subtypes of IIM with CAD was not uniformly observed. A population-based study conducted in Taiwan found a higher adjusted hazard ratio of CAD in polymyositis [3.73 (95% CI 2.83, 4.90)] than dermatomyositis [2.21 (95% CI 1.64, 2.99)] [16], which echoed our findings. On the other hand, another study found that dermatomyositis was associated with more atherosclerosis using flow-mediated dilation of brachial artery on ultrasound [17]. In our study, one possible explanation is that polymyositis patients might have more significant physical weakness than other subtypes of IIM like amyopathic dermatomyositis. In fact, the maximum CK and LDH levels were observed to be higher in obstructive CAD subgroup. Such physical inactivity might cause a higher risk of cardiovascular comorbidities and atherosclerosis, similar to the sedentary lifestyle observed in studies in general population [18] or patients with SLE [19].

Only diabetes mellitus was found to be independently associated with obstructive CAD with a high adjusted odds ratio. FG and HbA1c were also associated with obstructive CAD in the unadjusted analysis, highlighting the importance of screening and adequate glycemic control [20]. Regarding the association between systemic steroid and CAD, this study could not show the relationship of dosage and duration of steroid with obstructive CAD as in other rheumatic disease [21]. Small sample size of our study was the likely explanation.

The study has several limitations. First, the small sample size in both IIM and control groups limited the analysis. Sensitivity analysis in different subgroups was not possible. Increasing the sample size will likely improve the power for detecting risk factors of CAD in IIM. Second, the background data on demographics and underlying cardiovascular comorbidities (e.g., smoking) in the control group were incomplete. Furthermore, the lack of biochemical parameters in the control group precludes the comparison of metabolic risk factors burden with IIM patients. Finally, disease activity assessments by validated scores were not performed.

CONCLUSION

IIM patients carried a substantial risk of subclinical CAD on CTCA. The risk appeared to be disproportionately high compared to controls, in concordance with other immune-mediated inflammatory diseases. Traditional

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cardiovascular risk factors were more common in IIM patients with obstructive CAD, warranting regular monitoring and early control to prevent morbidities or mortalities.

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CONFLICTS OF INTEREST

All authors have disclosed no conflicts of interest.

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REFERENCES

[1] Wong VT, So H, Lam TT, Yip RM. Myositis-specific autoantibodies and their clinical associations in idiopathic inflammatory myopathies. Acta Neurol Scand. 2021;143(2):131-9. doi:10.1111/ane.13331

[2] Dobloug GC, Svensson J, Lundberg IE, Holmqvist M. Mortality in idiopathic inflammatory myopathy: results from a Swedish nationwide population-based cohort study. Ann Rheum Dis. 2018;77(1):40-7. doi:10.1136/annrheumdis-2017-211402

[3] Yang X, Hao Y, Zhang X, et al. Mortality of Chinese patients with polymyositis and dermatomyositis. Clin Rheumatol. 2020;39(5):1569-79. doi:10.1007/s10067-019-04910-w

[4] So H, Yip ML, Wong AK. Prevalence and associated factors of reduced bone mineral density in patients with idiopathic inflammatory myopathies. Int J Rheum Dis. 2016;19(5):521-8. doi:10.1111/1756-185X.12405

[5] Limaye V, Hakendorf P, Woodman RJ, Blumbergs P, Roberts-Thomson P. Mortality and its predominant causes in a large cohort of patients with biopsy-determined inflammatory myositis. Intern Med J. 2012;42(2):191-8. doi:10.1111/j.1445-5994.2010.02406.x

[6] Dobloug GC, Garen T, Brunborg C, Gran JT, Molberg Ø. Survival and cancer risk in an unselected and complete Norwegian idiopathic inflammatory myopathy cohort. Semin Arthritis Rheum. 2015;45(3):301-8. doi:10.1016/j.semarthrit.2015.06.005

[7] Schwartz T, Diederichsen LP, Lundberg IE, Sjaastad I, Sanner H. Cardiac involvement in adult and juvenile idiopathic inflammatory myopathies. RMD Open. 2016;2:e000291. doi:10.1136/rmdopen-2016-000291

[8] Diederichsen LP, Diederichsen AC, Simonsen JA, et al. Traditional cardiovascular risk factors and coronary artery calcification in adults with polymyositis and dermatomyositis: a Danish multicenter study. Arthritis Care Res (Hoboken). 2015;67(6):848-54. doi:10.1002/acr.22520

[9] Bohan A, Peter JB. Polymyositis and dermatomyositis (first of two parts). N Engl J Med. 1975;292(7):344-7. doi:10.1056/NEJM197502132920706

[10] So H, So J, Lam TT, et al. Seasonal effect on disease onset and presentation in anti-MDA5 positive dermatomyositis. Front Med (Lausanne). 2022 Feb 4;9:837024. doi:10.3389/fmed.2022.837024

[11] Lundberg IE, Tjärnlund A, Bottai M, et al. 2017 European League against Rheumatism/American College of Rheumatology classification criteria for adult and juvenile idiopathic inflammatory myopathies and their major subgroups. Arthritis Rheumatol. 2017;69(12):2271-82. doi:10.1002/art.40320

[12] Agatston AS, Janowitz WR, Hildner FJ, Zusmer NR, Viamonte M Jr., Detrano R. Quantification of coronary artery calcium using ultrafast computed tomography. J Am Coll Cardiol. 1990;15(4):827-32. doi:10.1016/0735-1097(90)90282-t

[13] Meng H, Lam SH, So H, Tam LS. Incidence and risk factors of major cardiovascular events in rheumatoid arthritis and psoriatic arthritis: a population-based cohort study. Semin Arthritis Rheum. 2024;65:152416. doi:10.1016/j.semarthrit.2024.152416

[14] Kariniemi S, Rantalaiho V, Virta LJ, Puolakka K, Sokka-Isler T, Elfving P. Multimorbidity among incident Finnish systemic lupus erythematosus patients during 2000-2017. Lupus. 2021;30(1):165-71. doi:10.1177/0961203320967102

[15] Lai EC, Huang YC, Liao TC, Weng MY. Premature coronary artery disease in patients with immune-mediated inflammatory disease: a population-based study. RMD Open. 2022;8(1):e001993. doi:10.1136/rmdopen-2021-001993

[16] Weng MY, Lai EC, Kao Yang YH. Increased risk of coronary heart disease among patients with idiopathic inflammatory myositis: a nationwide population study in Taiwan. Rheumatology (Oxford). 2019;58(11):1935-41. doi:10.1093/rheumatology/kez076

[17] Vincze M, Dér H, Kerekes G, et al. Decreased flow-mediated dilatation with increased arterial stiffness and thickness as early signs of atherosclerosis in polymyositis and dermatomyositis patients. Clin Rheumatol. 2014;33(11):1635-41. doi:10.1007/s10067-014-2561-y

[18] Dunstan DW, Barr EL, Healy GN, et al. Television viewing time and mortality: the Australian Diabetes, Obesity and Lifestyle Study (AusDiab). Circulation. 2010;121(3):384-91. doi:10.1161/CIRCULATIONAHA.109.894824

[19] Volkmann ER, Grossman JM, Sahakian LJ, et al. Low physical activity is associated with proinflammatory high-density lipoprotein and increased subclinical atherosclerosis in women with systemic lupus erythematosus. Arthritis Care Res (Hoboken). 2010;62(2):258-65. doi:10.1002/acr.20076

[20] Park C, Guallar E, Linton JA, et al. Fasting glucose level and the risk of incident atherosclerotic cardiovascular diseases. Diabetes Care. 2013;36(7):1988-93. doi:10.2337/dc12-1577

[21] So H, Lam TO, Meng H, Lam SHM, Tam LS. Time and dose-dependent effect of systemic glucocorticoids on major adverse cardiovascular event in patients with rheumatoid arthritis: a population-based study. Ann Rheum Dis. 2023;82(11):1387-93. doi:10.1136/ard-2023-224185