



Pericardial effusion in idiopathic inflammatory myopathies: A cross-sectional study from Asia and review of the literature

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Abstract

Objectives: Pericardial effusion is a rare clinical manifestation in idiopathic inflammatory myopathies (IIMs). It has been described in a small number of literature studies worldwide. We describe the clinical and laboratory characteristics of 19 IIM patients combined with pericardial effusion, and compare them with previously reported cases. The single-center observational-study-inspired collected of 156 IIM patients with complete data from January 1, 2016 to January 1, 2021 in the First Affiliated Hospital of Xinjiang Medical University, of which 19 patients had pericardial effusion.

Methods: The clinical characteristics of 19 IIM patients complicated with pericardial effusion were investigated by descriptive analysis and compared with previously reported cases.

Results: 19 cases of IIM patients had pericardial effusion (12.2%), patients without a large amount of pericardial effusion or pericardial tamponade. There was a predominance of women in the patients with 78.9% pericardial effusion. In the clinical examination, 10 cases showed chest tightness (52.6%), pulmonary fibrosis (47.4%), and the frequency of muscle nuclear magnetic, which suggested that muscle lymphocyte infiltration rate was 63.2%. Anti-Ro-52 antibody and anti-Jo-1 antibody were positive (26.3%, 42.1%). IIM patients with pericardial effusion were accompanied by decreased serum albumin levels and elevated ESR. In the literature review, the most common clinical characteristics of IIM patients with pericardial effusion were female, pulmonary fibrosis, shortness of breath, positive anti-Ro-52 pulmonary fibrosis, and anti-Jo-1 antibody.

Conclusion: In the study, 19 patients of IIMs with pericardial effusion present with chest tightness, and are accompanied by pulmonary fibrosis, positive anti-Jo-1 antibody, and anti-Ro-52 antibody. It is suggested that pericardial effusion in IIM patients may be related to anti-synthetase antibody.

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Keywords

Idiopathic inflammatory myopathies, pericardial effusion, pericarditis, clinical characteristics, China

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Introduction

Idiopathic inflammatory myopathies (IIMs) are a group of autoimmune diseases with proximal muscle weakness, rash, and myalgia as the main manifestations. It mainly includes clinical subtypes such as dermatomyositis, polymyositis, and amyopathic dermatomyositis.¹⁻⁵ The pathogenesis of IIM is still unclear, but a series of factors such as genetic susceptibility, environmental triggers, immunity, and medication history play a role in the development of the disease. IIMs may affect muscles, skin, joints, esophagus, lungs, heart, and other tissues and organs. Common complications of IIMs include interstitial lung disease, cancer, arrhythmias, and thrombosis, resulting in poor prognosis and increased mortality. The first symptoms are mostly rash and muscle weakness of proximal extremities.^{6,7}

Heart involvement is one of the common complications of IIMs, mainly manifested as left ventricular diastolic dysfunction, arrhythmia, and conduction block. Pericardial effusion is uncommon, and the clinical symptoms are often insidious, that is mostly discovered accidentally. Therefore, it is easy to be ignored by patients and doctors, leading to irreversible dysfunction and even death. Autoimmune disease is one of the causes of pericardial effusion.⁸⁻¹¹ There has been a lot of research on the clinical features of myositis with interstitial lung disease, cancers, and thrombosis. However, there were few studies on IIMs complicated with pericardial effusion at home and abroad. Therefore, this study analyzed the general data, clinical features, laboratory findings, and imaging features of IIMs combined with pericardial effusion, as well as, compared them with previously reported cases.

Materials and methods

Patients

This single-center observational-study-inspired was conducted in the First Affiliated Hospital of Xinjiang Medical University; using data collected from the health information base and patient anonymity, the subject which met the Helsinki declaration cannot be found. 156 patients with IIMs were collected from January 1, 2016 to January 1, 2021, which had complete data.

According to the diagnostic criteria for IIMs and echocardiography results, which were assessed by two experienced clinicians, 19 patients met the study criteria.

The diagnosis met the Bohan/Peter criteria⁸ and ENMC (European Neuromuscular Center) criteria.¹² Pericardial effusion was assessed by echocardiography.

Data

Clinical data were obtained through a retrospective review of medical records. The general information of the patients was recorded, including gender, ethnicity, age, age of first episode, course of disease, blood pressure, and body mass index (BMI); clinical manifestations, such as sun rash, Gottron sign, Gower sign, mechanic hand, erythema of the nail, V-sign, and muscle strength; the patient's laboratory test results when visiting our hospital, including blood routine, biochemical analysis, coagulation analysis, inflammatory factors, and erythrocyte sedimentation rate; immunological indicators: (1). Antibodies related to IIMs, including anti-RO-52 antibody and anti-polyphotic/hard skin (PM/SCL) 75/100 antibody; (2). Anti-histidine-specific antibody, antiodamyl TRNA synthetase (Jo-1) antibody, anti-threonyl TRNA synthetase (PL-7) antibody, antanetyl TRNA synthetase (PL-12) antibody, anti-glycyl TRNA synthetase (EJ) antibody, anti-hemineyl TRNA synthase (OJ) antibody, anti-melanoma differentiation related gene 5 (MDA5) antibody, and anti-nuclear matrix protein 2NXP2) antibody; Imaging examination and others: heart ultrasonic heart map, neck vascular ultrasound, muscle nuclear magnetic, high resolution CT etc. We collected all data that met the inclusion and exclusion criteria in the past 5 years for comprehensive analysis. The data were checked by two researchers to ensure accuracy. An experienced clinician was invited to assist in the assessment to address potential biases.

Ethics

With the approval of the Ethics Committee of First Affiliated Hospital of Xinjiang Medical University (K202110-01), we conducted the retrospective study that met the Helsinki Declaration.

Literature review

We searched the English-language literature in the PubMed database and EMBASE (Library of Xinjiang Medical University) for all related articles, using the following four

key words: idiopathic inflammatory myopathies, IIMs, pericarditis, and pericardial effusion.

Statistical analysis

SPSS26.0 statistical software was used. Data in line with normal distribution used average \pm standard deviation, and data that did not meet normal distribution were indicated by the median and percentile.

Results

Clinical and antibodies features of 19 idiopathic inflammatory myopathies patients with pericardial effusion

The clinical data of 156 patients with IIMs were collected in this study, among which only 19 patients had pericardial effusion, accounting for 12.2%. There were no patients with massive pericardial effusion or cardiac tamponade. The average age of the patients with pericardial effusion was 51 years old. We mainly studied the clinical manifestations and examination results of patients with IIMs complicated with pericardial effusion. There was a

predominance of women in the patients with pericardial effusion ($n = 15$, 78.9%). In the clinical examination, 8 cases showed pulmonary auscultation crackles in patients with pericardial effusion ($n = 8$, 42.1%), 5 cases had limb edema ($n = 5$, 26.3%), 10 cases showed chest tightness ($n = 10$, 52.6%), and 9 cases showed pulmonary fibrosis (47.4%). The frequency of muscle nuclear magnetic, which suggested muscle lymphocyte infiltration rate, was 63.2%. As it is shown in Table 1, there were five patients with pericardial effusion that showed anti-Jo-1 antibody was positive (26.3%). And eight patients with pericardial effusion showed that anti-Ro-52 antibody was positive (42.1%). In addition to the antibodies mentioned above, the following antibodies were positive: SSA (15.8%), MDA-5 (10.5%), SRP (5.3%), EJ (5.3%), PL-7 (5.3%), NXP2 (5.3%), PM-scl (10.5%), and AMA-M2 (15.8%). (Table 1)

Laboratory characteristics of IIM patients combined with pericardial effusion

Red blood cell distribution width (RDW) and cytotoxic T lymphocyte level (CTL) in IIM patients with pericardial effusion were higher, the albumin (ALB) average was little

Table 1. Clinical and antibodies features of 19 idiopathic inflammatory myopathies patients with pericardial effusion.

Patient	Age/ Sex	DPS	SPS	BMI	WR	CS	LD	CT	JT	Expectoration	AEMG	MNMRA	PF	PW	IJVP	Ab
1	71/M	125	70	20	0	No	Yes	No	No	No	YES	YES	No	YES	YES	Ro-52, SSA, Jo-1
2	59/F	130	80	23	0	Yes	No	No	No	YES	No	No	YES	No	YES	Ro-52, Jo-1
3	33/M	115	70	22	7	No	No	No	Yes	No	YES	YES	No	No	No	MDA-5
4	57/F	133	71	20	0	No	No	Yes	No	No	YES	No	YES	YES	No	Jo-1
5	50/F	129	89	22	0	No	No	Yes	Yes	YES	YES	No	No	No	No	Ro-52, Jo-1
6	29/F	91	61	24	10	No	No	No	No	No	YES	YES	No	No	No	EJ, PM-Scl
7	33/F	115	68	26	10	Yes	No	Yes	No	No	YES	YES	YES	YES	No	—
8	64/F	129	70	24	4	Yes	No	Yes	No	YES	No	No	YES	No	No	NXP2, PM-Scl, AMA-M2
9	64/F	115	52	27	0	Yes	Yes	Yes	No	No	No	YES	YES	No	YES	SSA, SRP
10	44/F	120	77	23	0	Yes	Yes	Yes	Yes	No	No	No	YES	YES	No	AMA-M2
11	51/M	106	63	20	0	No	Yes	Yes	No	No	YES	YES	No	YES	No	Ro-52
12	45/F	118	74	22	0	Yes	No	No	No	No	YES	YES	YES	No	YES	Ro-52, PL-7
13	51/F	116	83	20	12	Yes	No	No	No	YES	YES	YES	No	No	No	Ro-52
14	69/F	114	70	24	0	No	No	Yes	No	YES	No	No	YES	YES	YES	Ro-52, AMA-M2
15	49/F	95	53	13	0	No	No	Yes	No	No	No	YES	No	No	No	Jo-1
16	6/M	137	82	22	0	No	Yes	No	Yes	No	YES	YES	No	No	No	—
17	67/F	126	92	30	0	No	No	No	No	YES	YES	YES	No	YES	No	—
18	56/F	120	70	25	0	No	No	No	No	No	No	YES	YES	No	YES	Ro-52, MDA-5
19	60/F	136	94	26	0	Yes	No	Yes	No	YES	No	No	No	No	No	SSA

Abbreviations: Ab = antibodies, DPS = Diastolic pressure, SPS = Shrink pressure, BMI = Body Mass Index, WR = Weight reduction (Kg), CS = Crackling sound, LD = Limb edema, CT = Chest tightness holding breath, JT = Joint tenderness, AEMG = Abnormal EMG, MNMRA = Muscle NMR abnormalities, PF = Pulmonary fibrosis, PW = Progressive weakness, IJVP = Internal jugular vascular plaque, AMA-M2, Mitochondrial M2; EJ, Glycyl-tRNA-synthetase; Jo-1, Histidyl-tRNA-synthetase; MDA-5, Melanoma differentiation-associated gene five; SRP, Signal recognition particle; NXP2, Nuclear matrix protein 2; OJ, Isoleucyl-tRNA-synthetase; PM, Polymyositis; PL-7, Threonyl-tRNA-synthetase; PL-12, Alanlyl-tRNA-synthetase; SSA, Sjögren syndrome-A; TNF, Tumor necrosis factor.

lower (34.36 g/L). The median LDH was 389.14 U/L, the median BNP was 94.5 pg/mL, the mean D-dimer was 441 ng/mL, and the mean ESR was 35.44 mm/h in patients with pericardial effusion. (Table 2, Table 3)

Literature review

We found a total of 25 IIM patients with pericardial effusion in eight articles, of which 5 were case reports and three were retrospective articles. Based on these literature studies, we could only obtain the specific number of cases of IIM patients with pericardial effusion, but not the specific number of patients with clinical features such as pulmonary fibrosis and elevated CRP. Through comprehensive analysis of the full texts, we draw the conclusions: The main clinical manifestations in the overall group of 25 patients (present cohort and reported cases) were pulmonary fibrosis, positive anti-Ro-52 antibody, and positive anti-Jo-1 antibody. (Table 4)

Discussion

The heart involvement is the most important cause of death in patients with IIMs, such patients may experience heart conduction block, supraventricular arrhythmia, or cardiomyopathy, which may cause syncope, congestive heart failure, etc. Some studies have suggested that autoimmune diseases can cause pericardial effusion, which is a sub-clinical heart damage in patients with IIMs, this may be related to the decline of right heart function, but it cannot be ruled out that it is a chronic non-suppurative inflammatory reaction in which a large number of inflammatory cells infiltrate the primary disease.^{10,11,21,22} Among them, IIMs with pericardial effusion is easy to be ignored in clinical practice, except that pericardial effusion caused by autoimmune diseases is usually small in volume and hemodynamics are not significant, it is also considered that this is related to the lower incidence of IIMs itself.¹³

In this study, we analyzed the clinical and laboratory characteristics of patients with IIMs complicated with pericardial effusion at First Affiliated Hospital of Xinjiang Medical University over a 5-year period. There are few cases of IIMs with pericardial effusion. Due to the small sample size, some clinical features may have been over-amplified, so we also included a review of published cases in the literature to overcome these problems. Studies have suggested that the frequency of IIMs with pericardial effusion is 12.37%–17%,²³ which is similar to the 12.2% in this study, and similar to other studies, the patients with pericardial effusion in our collected cases are all small or moderate pericardial effusions (small effusion is 50–100 mL, moderate effusion is 100–500 mL, and large effusion > 500 mL); however, if there is no large amount of

Table 2. Laboratory test results of patients with IIMs.

Characteristic	Patient
WBC ($10^9/L$)	7.15 ± 2.67
N ($10^9/L$)	5.43 ± 2.46
LY ($10^9/L$)	1.12 ± 0.44
MO ($10^9/L$)	0.45 ± 0.23
RBC ($10^{12}/L$)	4.47 ± 0.52
HGB (g/L)	121.74 ± 21.03
RDW (%)	14.98 ± 1.68
PLT ($10^9/L$)	278.47 ± 130.91
K (mmol/L)	3.86 ± 0.54
NA (mmol/L)	138.56 ± 4.25
CL (mmol/L)	102.89 ± 3.49
CA (mmol/L)	2.21 ± 0.18
BUN (mmol/L)	5.80 ± 2.38
Cr (mmol/L)	35.01 ± 12.56
TG (mmol/L)	2.20 ± 1.11
CHO (mmol/L)	4.09 ± 1.52
HDL-C (mmol/L)	1.03 ± 0.35
LDL-C (mmol/L)	2.57 [1.48,3.97]
T-BIL ($\mu\text{mol/L}$)	9.47 ± 4.56
ALB (g/L)	34.36 ± 4.97
GLB (g/L)	31.59 ± 7.50
ALP (U/L)	71 [59.90,91]
AST (U/L)	53.94 [22.70,243.50]
ALT (U/L)	79.20 [16.70,211.68]
LDH (U/L)	389.14 [314.67,805.63]
DRJT (ng/mL)	441 [237.75,634]
25-VD3 (nmol/L)	36.73 ± 22.98
AFP (ng/mL)	3.03 ± 1.15
CEA (ng/mL)	2.15 [.09,3.95]

Table 3. Connective tissue disease associated markers.

Characteristic	Patient
CK (ng/ml)	249.90 [32.42,3032.50]
CK-MB (IU/L)	13.35 [1.59,129.62]
ESR (mm/h)	35.44 ± 18.47
IL-6 (pg/ml)	10.44 [1.50,34.09]
CRP (mg/L)	6.35 [3.15,17.88]
BNP (pg/mL)	94.50 [41.20,444.27]
PCT (ng/mL)	0.07 [0.02,0.13]
C3 (g/L)	0.85 ± 0.16
C4 (g/L)	0.18 ± 0.06
IgG (g/L)	15.62 ± 5.50
IGA (g/L)	2.53 ± 1.12
IgM (g/L)	1.23 [1.09,1.84]
Th (%)	34.19 ± 13.36
CTL (%)	37.08 ± 13.36
TC (%)	67 [30.28,74.38]
BC (%)	16.38 ± 10.23

Table 4. Main characteristics of patients with pericardial effusion/pericarditis, previous and present reports.

First author, ref	Number of patients	With pericardial effusion/pericarditis	Main characteristics of patients with pericardial effusion/pericarditis
Li ¹³	271	10	Pulmonary fibrosis, skin damage, anemia, elevated creatine kinase, decreased C3 and serum albumin, positive anti-Ro-52 antibody, and Jo-1 antibody
Mediavilla ¹⁴	2	2	Pulmonary fibrosis, Raynaud's phenomena, arthritis, tenosynovitis
Shah ¹⁵	1	1	Pulmonary fibrosis, anti-synthetase syndrome, shortness of breath, cough, more prevalent in women, positive Jo-1 antibody, PL-7, PL-12, E], O] and SRP.
Byrnes ¹⁶	13	1	Woman
Luo ¹⁷	96	8	Older age (54.58 ± 10.58) years, pulmonary fibrosis, elevated C-reactive protein [13.55 (8.96, 38.35) mg/L], lactate dehydrogenase [408.0 (255.0, 587.0) U/L]. Positive Ro-52 antibody and elevated LDH.
Krajnc ¹⁸	1	1	A 53-year-old woman, pulmonary fibrosis, polyarthritis, Raynaud's syndrome, carpal tunnel syndrome, sclerodactylia, and positive Jo-1-antibodies
Hayashi ¹⁹	1	1	A 69-year-old man, pulmonary fibrosis, neck muscle weakness, symmetric proximal muscle weakness, skin rash and elevated creatine kinase, positive E], pulmonary fibrosis, and anti-synthetase syndrome
Cheston ²⁰	1	1	A woman, chest pain, shortness of breath, arthralgia, muscle weakness, and skin discolouration

pericardial effusion or pericardial tamponade,^{7,24–27} it is more reliable.

In our current research, nine of the patients with pericardial effusion had pulmonary fibrosis (47.4%) and presented pulmonary crackles on auscultation (42.1%). Some case reports showed that patients with pericardial effusion have rapidly progressive interstitial lung disease, or they came with each other.^{13–15,28} Crackling sounds are a common sign of interstitial lung disease, it is considered to be related to the severity of interstitial lung disease and the sample size.

Anti-Jo-1 antibody was significantly related to pulmonary hypertension, which could cause lung damage and heart damage, anti-Jo-1 antibody could also act as a chemotactic factor to induce cell damage mediated by T cells,¹³ at the same time, pericardial effusion would compress the surrounding lung tissues, affected the gas exchange of the respiratory system, and reduced the cardiac output, which could cause chest tightness, shortness of breath, edema, and other symptoms in patients. In this study, among the 19 patients with pericardial effusion, 26.3% had positive Jo-1 antibody, while about 8.8% of the patients without pericardial effusion had positive Jo-1 antibody. Also, patients with pericardial effusion had a significantly higher proportion of chest tightness compared with patients without pericardial effusion. A multicenter study of 18 patients followed for 2.8–10.7 years suggested pericardial effusion as a possible manifestation of association with anti-PL-7 antibodies.²⁹ There were 25 IIM samples in our study, and there was one pl-7 positive sample in both

groups ($p = 0.100$), considering the reasons of insufficient sample size and short follow-up time.

In addition, IIMs combined with pericardial effusion can cause muscle inflammatory infiltration and decreased serum albumin (34.36 g/L), which could lead to limb edema,³⁰ at the same time, tissue and organ damage was related to the activity of IIMs, and the disease activity of IIMs depended largely on the intensity of the inflammatory response, and some studies have shown that serum albumin is negatively correlated with indicators representing IIMs activity,^{31,32} which was similar to this study in the combined pericardial effusion group; ALB was reduced in the muscle NMR results, patients in the group with pericardial effusion had a higher proportion of muscle lymphocyte infiltration.

There were few cases of IIMs complicated with pericardial effusion, and few studies have focused on the clinical characteristics and auxiliary examination characteristics of these patients. We used the four keywords: idiopathic inflammatory myopathies, IIMs, pericarditis, and pericardial effusion, to search in two English databases (PubMed and EMBASE), and eight literatures met the requirements. They included 1 (1/13, 7.7%), 8 (8/96, 8.3%), and 10 (10/271, 3.7%) patients, additionally adding five case reports, for a total of 25 patients.^{13–20} Anti-synthetase syndrome is described classically by the triad of interstitial lung disease, inflammatory myositis, and presence of aminoacyl-tRNA synthetase antibodies. In common with other reported IIM patients with pericardial effusion more prevalent in women, pulmonary fibrosis and positive anti-Jo-1 antibody were the most frequent

manifestations, and these common features were confirmed in a review of all cases published to date in the literature. It is suggested that pericardial effusion in IIM patients may be related to anti-synthetase antibody. In addition, muscle weakness, elevated creatine kinase, positive anti-Ro-52 antibody, and PL-7 were also present in a subset of patients.

The study also had several limitations: First, there was a lack of extensive literature and guidelines on pericardial effusion associated with idiopathic inflammatory myopathy. Second, it was a retrospective observational-study-inspired. Further prospective studies were needed to verify the conclusions of this study. Next, this study did not carry out the calculation and demonstration of the selected sample size, but we collected of all IIM patients with complete data from January 1, 2016 to January 1, 2021 in the First Affiliated Hospital of Xinjiang Medical University. Because IIMs were a rare disease with few cases to look for, the data available may not be comprehensive and may result in bias for a more complete assessment of the disease. In the future, we would include a larger number of samples from multiple regions to further explore the clinical manifestations and epidemiological characteristics of pericardial effusion in IIMs.

Conclusion

In summary, like other reported IIM patients with pericardial effusion, most of the IIM patients with pericardial effusion were female, and complicated with chest tightness, pulmonary fibrosis, positive anti-Jo-1 antibody, and anti-Ro-52 antibody. It is suggested that pericardial effusion in IIM patients may be related to anti-synthetase antibody and anti-Ro-52 antibody, which needs to be confirmed by our future studies.

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Ethical approval

Ethical approval for this study was obtained from *NAME OF ETHICS COMMITTEE OR INSTITUTIONAL REVIEW BOARD (APPROVAL NUMBER/ID)*. With the approval of the

Ethics Committee of First Affiliated Hospital of Xinjiang Medical University (K202110-01), we conducted the retrospective study that met the Helsinki Declaration.

Informed consent

Informed consent was not sought for the present study because *REASON*. We performed a retrospective study, collecting data from the health information base, and did not involve any prospective collection of data or patient interventions. The patient is anonymous, the subject cannot be found, the rights and welfare of the subject will not be adversely affected, and the research project does not involve commercial interests. So, we applied to exempt informed consent for study participants. Our research followed the guidelines outlined in the Declaration of Helsinki.

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References

1. Mainetti C, Terziroli Beretta-Piccoli B and Selmi C (2017) Cutaneous manifestations of dermatomyositis: a comprehensive review. *Clin Rev Allergy Immunol* 53(3): 337–356.
2. Ge Y, Lu X, Shu X, Peng Q and Wang G (2017) Clinical characteristics of anti-SAE antibodies in Chinese patients with dermatomyositis in comparison with different patient cohorts. *Sci Rep* 7(1): 188.
3. Dalakas MC (2020) Inflammatory myopathies: update on diagnosis, pathogenesis and therapies, and COVID-19-related implications. *Acta Myol* 39(4): 289–301.
4. Tanboon J, Uruha A, Stenzel W and Nishino I (2020) Where are we moving in the classification of idiopathic inflammatory myopathies? *Curr Opin Neurol* 33(5): 590–603.
5. Leclair V, Notarnicola A, Vencovsky J and Lundberg IE (2021) Polymyositis: does it really exist as a distinct clinical subset? *Curr Opin Rheumatol* 33(6): 537–543.
6. Li Y, Wang P, Li L, Wang F and Liu Y (2018) Increased risk of venous thromboembolism associated with polymyositis and dermatomyositis: a meta-analysis. *Ther Clin Risk Manag* 14: 157–165.
7. Yang SH, Chang C and Lian ZX (2019) Polymyositis and dermatomyositis - challenges in diagnosis and management. *J Transl Autoimmun* 2: 100018.
8. Liu XH, Feng XJ, Shi JY, et al. (2020) The quest for diagnostic approaches of cardiac involvement in polymyositis and dermatomyositis. *Ann Palliat Med* 9(4): 2256–2270.
9. Vencovský J, Alexanderson H and Lundberg IE (2019) Idiopathic inflammatory myopathies. *Rheum Dis Clin North Am* 45(4): 569–581.
10. Zhang L, Wang GC, Ma L and Zu N (2012) Cardiac involvement in adult polymyositis or dermatomyositis: a systematic review. *Clin Cardiol* 35(11): 686–691.

11. Lazaros G, Vlachopoulos C, Lazarou E, Tousoulis D and Tsioufis C (2021) Contemporary management of pericardial effusion. *Panminerva Med* 63(3): 288–300.
12. Bohan A and Peter JB (1975) Polymyositis and dermatomyositis (first of two parts). *N Engl J Med* 292(7): 344–347.
13. Li YH, Du Y, Li L, et al. (2020) Analysis of immunological characteristics of dermatomyositis patients with myocardial involvement. *Zhonghua Yixue Zazhi* 100(10): 753–756.
14. Mediavilla JD, Giner P, López-Gómez M, Matarán L, Ibáñez MA and Jiménez-Alonso J (1992) Comments with respect to 2 patients with anti-Jo 1 antibodies. *An Med Interna* 9(8): 389–390.
15. Shah A and Patel SR (2016) Acute onset anti-synthetase syndrome with pericardial effusion and non-specific interstitial pneumonia. *J Clin Med Res* 8(9): 683–687.
16. Byrnes TJ, Baethge BA and Wolf RE (1991) Noninvasive cardiovascular studies in patients with inflammatory myopathy. *Angiology* 42: 843–848.
17. Luo L, Xing XY, Xiao YS, et al. (2021) Clinical and immunological characteristics of patients with anti-synthetase syndrome complicated with cardiac involvement. *Beijing Da Xue Bao Yi Xue Ban* 53(6): 1078–1082.
18. Krajnc I (1995) Anti-Jo-1-positive polymyositis with interstitial alveolitis and pericardial effusion. Overview of 3 years treatment. *Wien Klin Wochenschr* 107(19): 590–592.
19. Hayashi K, Machida Y, Katayama Y, et al. (2019) A case of antisynthetase syndrome with anti-EJ antibody complicated by pericarditis. *Rinsho Shinkeigaku* 59(1): 21–26.
20. Cheston HJ, Akhoun C, Roy SD and Reynolds P (2022) Managing myocarditis in a patient with newly diagnosed dermatomyositis. *BMJ Case Rep* 15(3): e246989.
21. Rider LG, Aggarwal R and Machado PM (2018) Update on outcome assessment in myositis. *Nat Rev Rheumatol* 14(5): 303–318.
22. Lazaros G, Antonopoulos AS, Imazio M, et al. (2019) Clinical significance of pleural effusions and association with outcome in patients hospitalized with a first episode of acute pericarditis. *Intern Emerg Med* 14(5): 745–751.
23. Albrecht K, Huscher D, Callhoff J, et al. (2020) Trends in idiopathic inflammatory myopathies: cross-sectional data from the German National Database. *Rheumatol Int* 40(10): 1639–1647.
24. Mocan M, Mocan Hognogi LD, Anton FP, et al. (2019) Biomarkers of inflammation in left ventricular diastolic dysfunction. *Dis Markers* 2019: 7583690.
25. Acosta I, Matamala JM, Jara P, Pino F, Gallardo A and Verdugo R (2019) Idiopathic inflammatory myopathies. A review. *Rev Med Chil* 147(3): 342–355.
26. Xu L, Yan J, Zhang F, et al. (2021) Use of inflammatory biomarkers and real-time cardiac catheterisation to evaluate the left ventricular diastolic function in patients with diastolic Heart Failure. *Heart Lung Circ* 30: 396–403.
27. Hanson MG and Chan B (2021) The role of point-of-care ultrasound in the diagnosis of pericardial effusion: a single academic center retrospective study. *Ultrasound J* 13(1): 2.
28. Badui E, Mintz G and Robles E (1986) The heart in dermatomyositis and polymyositis. *Arch Inst Cardiol Mex* 56(1): 71–76.
29. Labirua-Iturburu A, Selva-O’Callaghan A, Vincze M, et al. (2012) Anti-PL-7 (anti-threonyl-tRNA synthetase) anti-synthetase syndrome. *Medicine* 91(4): 206–211.
30. Duchesne M, Leonard-Louis S, Landon-Cardinal O, et al. (2020) Edematous myositis: a clinical presentation first suggesting dermatomyositis diagnosis. *Brain Pathol* 30(5): 867–876.
31. Gao H, Zhang XW, He J, et al. (2018) Prevalence, risk factors, and prognosis of interstitial lung disease in a large cohort of Chinese primary Sjogren syndrome patients: a case-control study. *Medicine (Baltim)* 97(24): e11003.
32. Gao MZ, Huang YL, Wu XD, et al. (2018) Red blood cell distribution width and neutrophil to lymphocyte ratio are correlated with disease activity of dermatomyositis and polymyositis. *J Clin Lab Anal* 32(1): e22209.