Objectives: To evaluate clinical utility of perfusion and myocardial fatty acid metabolism mismatch in SSc patients without cardiac failure.

Methods: All patients who visited St. Marianna University Hospital from 2009 to 2015 and performed cardiac scintigraphy using ^{99m}Tl and ¹²³I-BMIPP were retrospectively evaluated. Patients who fulfilled American Collage of Rheumatology classification criteria of SSc and systemic lupus erythematosus (SLE) were selected. We subtracted %uptake of metabolism (¹²³I-BMIPP) from that of perfusion (^{99m}Tl) on each 17 myocardial segments standardized by American Heart Association. We compared sum of all the scores and each score in 3 coronary artery regions between patients with SSc and SLE. Furthermore, we evaluated incidence of cardiac death or cardiac failure depending on the scores of each coronary artery regions.

Results: Among 177 patients, we analyzed the data in 22 cases with SSc and 23 with SLE. The sum of all mismatch scores was not significantly different between the 2 groups (p=0.37). The mismatch score of left anterior descending coronary artery (LAD) region was significantly higher in SSc than SLE (p=0.05) (Figure 1A). We next divided SSc patients into 2 groups depending on degree of the LAD mismatch score and found the group with low score had higher incidence of cardiac death or cardiac failure (p=0.04) than that with high score (Figure 1B).

Figure 1

Conclusions: The low mismatch score in LAD region of cardiac scintigraphy could be associated with cardiac death or cardiac failure in SSc patients. Since myocardial fibrosis might replace viable cardiomyocytes leading to loss of ventricular volume, metabolic defect by scintigraphy might be less detectable.

Disclosure of Interest: None declared

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FRI0392 OPPORTUNISTIC INFECTIONS IN PATIENTS WITH MYOSITIS. A RETROSPECTIVE COHORT STUDY

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Background: Idiopathic inflammatory myopathies, also known as myositis, are a heterogeneous group of acquired diseases of probable autoimmune origin, characterized by the presence of inflammatory muscle infiltrates. Infectious complications are not uncommon in myositis, and some predisposing factors, such as upper esophageal involvement, calcinosis cutis, ventilatory insufficiency due to diaphragm involvement, and immunosuppressive drug therapies, seem to be implicated.

Objectives: To describe the prevalence, clinical characteristics, and risk factors for opportunistic infection (OI) in a cohort of patients with inflammatory myopathies, and compare mortality rates between those with and without OIs.

Methods: In total, 204 patients from our myositis cohort were reviewed to identify patients who had experienced an OI during the period 1986–2014. The patients' clinical characteristics, treatment received, and outcome were systematically recorded. Disease activity at the OI diagnosis and cumulative doses of immunosuppressive drugs were analyzed, as well as the specific pathogens involved and affected organs.

Results: The prevalence of OI in the total cohort, was 6.4%: viruses, 44.4%

(varicella-zoster virus, cytomegalovirus); bacteria, 22.2% (*Salmonella* sp., *Mycobacterium tuberculosis*, *M. chelonae*); fungi, 16.7% (*Candida albicans*, *Pneumocystis jirovecii*); and parasites, 16.7% (*Toxoplasma gondii*, *Leishmania* spp.) were the pathogens detected. Lung and skin/soft tissues were the organs most commonly affected (27.8%). Overall, 55.6% of OIs developed during the first year after the myositis diagnosis and were significantly associated with administration of high-dose glucocorticoids (p=0.0148). Fever at onset of myositis (p=0.0317), biological therapy (p<0.001) and sequential administration of 4 or more immunosuppressive agents during myositis evolution (p=0.0032) were significantly associated with OI. All-cause mortality in the OI group was 3.69 deaths per 100 patients/year versus 3.40 in the remainder of the cohort (p=0.996).

Conclusions: The prevalence of OI was 6.4% in our myositis cohort. High-dose glucocorticoids at disease onset and severe immunosuppression are implicated in the development of these complications. Mortality did not differ from the remainder of the cohort.

Disclosure of Interest: None declared

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FRI0393 PREVALENCE OF MYOSITIS-SPECIFIC ANTIBODIES IN IDIOPATHIC INFLAMMATORY MYOPATHY COMPARED TO DISEASE AND HEALTHY CONTROLS

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Background: Myositis-specific autoantibodies (MSA) are increasingly recognized as important diagnostic and prognostic markers in idiopathic inflammatory myopathies (IIM) (polymyositis, dermatomyositis, sporadic inclusion body myositis and necrotizing autoimmune myositis). The prevalence of these MSAs in other systemic autoimmune rheumatic diseases and neuromuscular diseases is unclear.

Objectives: To compare positivity of MSA in a cohort of IIM patients to positivity in healthy controls and different systemic autoimmune rheumatic diseases (SARD) or chronic inflammatory demyelinating polyneuropathy (CIDP).

Methods: A line immunoassay (Myositis 12 IgG DOT for BlueDiver) for IgG autoantibodies against Jo-1, PL-7, PL-12, EJ, SRP, Mi-2, MDA-5, TIF1-γ, HMGR, SSA/Ro52kD, SAE1/2 and NXP-2 antigens was performed in patients with IIM (n=146), healthy controls (blood donors, n=40) and disease controls (n=200). The disease control group consisted of patients with other SARD (rheumatoid arthritis, RA; systemic sclerosis, Ssc; Sjögren's syndrome, SjS; and systemic lupus erythematosus, SLE) (n=40 for each disease group) and CIDP (n=40). A result >10 arbitrary units was considered significantly positive.

Results: 50% of 146 patients with IIM tested positive for an MSA (table 1), compared to 3.5% of 200 disease controls (table 1). 1 SSc patient was positive for Jo-1, 1 CIDP patient was positive for PL12, 2 SSc patients were positive for TIF1-gamma, 2 patients (1 SSc and 1 SjS patient) were positive for SAE1/2, and 1 SjS patient was positive for NXP2. The prevalence of SAE1/2 and TIF1-gamma positivity was similar in the IIM and disease control group. No healthy control had a significantly positive MSA.

Antibody detected	IIM (n=146)	Disease controls (n=200)	Healthy controls (n=40)
1 positive (>10 AU) MSA detected	68 (47%)	7 (3.5%)	0 (0%)
Jo-1	28 (19%)	1 (0.4%)	0 (0%)
PL12	3 (2%)	1 (0.4%)	0 (0%)
PL7	1 (1%)	0 (0%)	0 (0%)
EJ	2 (1%)	0 (0%)	0 (0%)
Mi-2	7 (5%)	0 (0%)	0 (0%)
MDA-5	7 (5%)	0 (0%)	0 (0%)
SAE1/2	3 (2%)	2 (0.8%)	0 (0%)
TIF1-gamma	2 (1%)	2 (0.8%)	0 (0%)
NXP2	5 (3%)	1 (0.4%)	0 (0%)
SRP	3 (2%)	0 (0%)	0 (0%)
HMGR	7 (5%)	0 (0%)	0 (0%)
>1 positive (>10 AU) MSA detected	4 (3%)	0 (0%)	0 (0%)
Jo-1 + EJ	1		
Jo-1 + NXP2	1		
SAE1/2 + NXP2	1		
Jo-1 + SAE1/2 + NXP2	1		

Conclusions: MSA positivity in patients with a clinical non-IIM diagnosis (other SARD or CIDP) is infrequent compared to positivity in the IIM group. For TIF1-gamma and SAE1/2 assay performance may need to be optimized. The distribution of subtypes of MSA in this IIM cohort is consistent with data of previous studies.¹

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