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THU0328

DO PATIENTS WITH SYSTEMIC SCLEROSIS HAVE ULTRASONOGRAPHIC MODIFICATIONS OF SALIVARY GLANDS SUGGESTIVE OF SJOGREN SYNDROM?

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Background: Modifications in ultrasonographic aspect of major salivary glands have been reported in patients with primary Sjogren Syndrom (pSS) with good diagnostic accuracy. Sicca symptoms are frequently observed in Systemic sclerosis (SSc).

Objectives: To assess the ultrasonographic echostructure of major salivary glands in patients with SSc and compare the modifications with those of patients with pSS or controls with sicca symptoms.

Methods: We performed a monocentre case-control study between 2014 and 2017 in the university hospital of Clermont-Ferrand (France). Patients with SSc and pSS were fulfilling the American College of Rheumatology/European League against Rheumatism (ACR/EULAR) 2013 and the ACR 2012 classification criteria respectively. Controls patients were complaining of sicca symptoms but did not meet the ACR 2012 criteria. Bilateral parotid and submandibular glands ultrasound (US) were performed in all patients by the same operator blinded to the diagnosis. Inhomogeneity of each of the 4 major salivary glands in B-Mode was graded using the Jousse-Joulin scoring system (scale of 0 to 4) as previously described.[1] The highest grade among the 4 glands was retained as suggestive of Sjogren Syndrom if ≥ 2 .

Results: A total of 108 patients were included: SSc (n=25), pSS (n=48) and controls (n=35). Among the 48 patients with pSS, 12 were receiving hydroxychloroquine, 4 an immunosuppressor, 77% had antinuclear antibodies at a significant level ($\geq 1/640$), 26 (54%) anti-SSA antibodies, 14 (29%) anti-SSB antibodies, 40/45 (89%) had a labial salivary biopsy suggestive of pSS (Chisholm and Mason score ≥ 3). Comparing the pSS and the control groups, performance of a US echostructure grade ≥ 2 for the diagnosis of pSS was good: Se=75%, Sp=91.4%, positive predictive value= 92.3%, negative predictive value= 72.7%.

Among the 25 SSc patients, 7 had immunosuppressor therapy, 8 had a localized SSc. Shimer's test ≤ 5 mm in 5 minutes was present in 9/18 (53%), unstimulated salivary flow ≤ 0.1 mL/minute in 8/14 (57.1%).

In the SSc group, 12 patients had an US echostructure grade 0, 6 grade 1, and 7 patients (28%) had an US echostructure grade of ≥ 2 : US score=3 (n=5), US score=4 (n=2). Anti-SSA antibodies were found in 1/7 patients with an US echostructure grade ≥ 2 and 2/18 patients with US echostructure grade 0 or 1.

	SSc group (n=25)	pSS group (n=48)	Control group (n=35)
Age, mean $\pm$ SD, years	64.8 $\pm$ 9.3	62.1 $\pm$ 12.3	55 $\pm$ 15
Female sex,%	88	96	97.2
Disease duration, mean $\pm$ SD, years,	16.7 $\pm$ 11.1	10.5 $\pm$ 7.7	Not applicable
US Score ≥ 2 , n (%)	7 (28)	36 (75)	3 (8.6)

Conclusion: Nearly one third of patients with SSc have US echostructure changes suggestive of Sjogren Syndrom regardless of the presence of anti-SSA antibodies.

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THU0329

RED FLAG SIGNS OF SYSTEMIC SCLEROSIS ARE PREVALENT IN SUBJECTS WITH RAYNAUD'S PHENOMENON IN THE GENERAL POPULATION AND MAY BE A PROXY FOR LUNG INVOLVEMENT

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Background: Pulmonary involvement in systemic sclerosis (SSc) is very difficult to treat when diagnosed too late. Therefore, in order to optimally use "the window of opportunity" more attention should be given to the early identification of SSc. To the best of our knowledge, no studies exist that have structurally assessed the epidemiology of red flag signs and potential signs of pulmonary involvement in patients with Raynaud's phenomenon (RP).

Objectives: To assess the prevalence of red flag signs in participants with RP. Moreover, we aim to investigate the occurrence of pulmonary signs and symptoms in participants with red flag signs.

Methods: We retrospectively analyzed data from the LifeLines Cohort Study, which is a large population-based cohort study in the Northern parts of the Netherlands. A total of 74011 participants completed the connective tissue disease questionnaire. The presence of RP and red flag signs for SSc (i.e., puffy fingers, skin thickening distal, skin thickening proximal, and pitting scars) were obtained. Patients were classified as having red flag signs by the presence of at least one red flag sign in addition to RP. In addition, patient characteristics, self-reported pulmonary complaints, spirometry (screening for interstitial lung disease (ILD)), and uric acid (global screening for pulmonary arterial hypertension (PAH)) were also obtained. Three groups of participants were formed, namely: participants with RP and red flag signs (n=981), participants with RP without red flag signs (n=2946), and participants without RP and without red flag signs (n=70037).

Results: The prevalence of red flag signs was 5 fold higher in participants with RP, as compared to the non-RP group (RP 25% [23.7-26.4], non-RP 5% [4.9-5.2], $p<0.001$). A total of 413(42.1%) of the participants with RP and red flag signs reported dyspnea, which was 1.5 fold higher than in those with RP but without red flag signs, and two-fold higher compared to participants without RP and without red flag signs ($p<0.001$). Moreover, dyspnea in rest and after exertion was more prevalent in participants with RP and red flag signs ($p<0.001$). In addition, participants with RP and red flag signs more frequently reported to suffer from pulmonary fibrosis ($p<0.001$, table 1), and had the lowest forced vital capacity, as compared to the other groups ($p<0.001$). Conversely, uric acid was not found to be elevated in participants with RP and red flag signs.

Conclusion: This unselected cohort study from the general population demonstrates that the prevalence of red flag signs in subjects with RP may be as high as 25%. Potential signs and symptoms of pulmonary complaints are more prevalent in participants with RP who also reported red flag signs. This could indicate an increased risk of pulmonary involvement (i.e., ILD and PAH) in RP patients with red flag signs, although additional specific tests are mandatory to substantiate definite disease.

Abstract THU0329 – Figure 1. Prevalence of red flag signs in participants with and without Raynaud's phenomenon.

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THU0330

PREVALENCE OF RAYNAUD'S PHENOMENON IN THE NORTHERN PARTS OF THE NETHERLANDS: AN EPIDEMIOLOGICAL STUDY OF THE LIFELINES COHORT

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Background: Although several previously conducted studies reported on the prevalence of Raynaud's phenomenon (RP) in different regions of the world, these studies often included a limited number of selected individuals. Moreover, no studies exist that have systematically assessed the relative contribution of known etiological factors of RP in the general population of the Netherlands.

Objectives: To assess the prevalence of RP, and gender-specific etiological factors associated with RP in the Northern parts of the Netherlands.

Methods: Data from the Lifelines cohort were analyzed, in which all participants completed the self-administered validated connective tissue disease questionnaire. Subjects who reported cold-sensitive fingers and bi- or triphasic colour changes in response to cold were considered to suffer from RP. Known etiological factors such as hormonal status, body mass index (BMI), smoking behaviour, and comorbidities were all assessed in a standardised way.

Results: In total 93935 participants completed the questionnaire (mean age 45.6 ±12.9). The prevalence of RP was 4.2% [95% CI 4.1-4.4] which was approximately three-fold higher in females (5.7%, 95% CI [5.5-5.9]), as compared to males (2.1%, 95% CI [1.9-2.2], p-value <0.001, figure 1). Regarding gender-specific risk factors associated with RP, we observed that BMI <18.5 (OR 4.6 [2.4-8.7], p<0.001), cardiovascular disease (OR 1.53 [95% CI 1.31-1.78], p<0.001), history of cancer (OR 1.40 [1.00-1.95], p=0.049), use of beta-blockers (OR 1.39 [1.06-1.83], p=0.01), and smoking (OR 1.28 [1.09-1.51], p=0.003) were associated with an increased odds of RP in men. Conversely, alcohol consumption, diabetes and age were not associated with RP in men. In females, BMI<18.5 (OR 2.9 [2.27-3.64], p<0.001), cardiovascular disease (OR 1.42 [1.32-1.54], p<0.001), receiving hormonal contraception (OR 1.17 [1.08-1.26], p<0.001), and hormonal replacement therapy (OR 1.14 [1.04-1.25], p=0.007) were associated with increased odds of RP. Moreover, smoking behavior, use of beta-blockers, alcohol consumption, and diabetes were not associated with RP in women. A BMI>30 was associated with a strongly decreased odds of RP in both men (OR 0.22 [0.11-0.42], p<0.001) and women (OR 0.35 [0.28-0.44], p<0.001).

Conclusion: This large cohort study found a prevalence of 4.2% of RP in the Northern part of the Netherlands, with an expected predominance in young female subjects. Moreover, the etiologic risk factors of RP are multifactorial and clearly gender-specific (e.g., hormonal status in women, smoking behavior and use of beta-blockers in men), with underweight strongly increasing and obesity strongly decreasing the likelihood of RP in both sexes. This might suggest that different mechanisms influence the expression of RP in men and women.

Abstract THU0330 – Figure 1. Prevalence of RP depicted for men and women.

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THU0331

SKELETAL TROPONIN I A POSSIBLE NOVEL BIOMARKER FOR MANAGEMENT OF PATIENTS WITH IDIOPATHIC INFLAMMATORY MYOPATHIES

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Background: The current biomarkers for the diagnosis and monitoring of diseased and injured skeletal muscles, such as creatine kinase (CK), have limited tissue specificity and incapability to differentiate between pathological and physiological changes. Thus, new biomarkers with improved diagnostic certainty are needed. Skeletal troponin I (skTnI) is a promising new biomarker for injured and diseased skeletal muscle tissue. Although studies have reported that circulating skTnI levels are elevated in response to trauma, exercise and various muscular diseases (1), its clinical utility to serve as a diagnostic indicator has largely been unexplored.

Objectives: Our aim was to develop and validate a novel assay for skTnI, and to assess its clinical performance with idiopathic inflammatory myopathy (IIM) patients.

Methods: A two-step fluoroimmunoassay was used to analyze the levels of skTnI in samples from healthy reference individuals (n=125), trauma patients (n=151), and patients with IIM (n=94). Later, skTnI and CK levels in patients with IIM were compared according to their disease activity status (active, pre-active or stable).

Results: The limit of detection was 1.2 ng/ml, and the upper reference limit (90th percentile) was 5.4 ng/ml. The median skTnI concentrations were <LoD, 2.7 ng/ml, and 9.8 ng/ml in reference, trauma, and IIM cohorts, respectively. Differences in measured skTnI levels were statistically significant between all three study cohorts (Mann-Whitney p<0.001 for all). skTnI and CK had a strong positive correlation (Spearman's r=0.848, p<0.001). With skTnI, patients in both pre-active and active IIM were differentiated from stable phase patients (33.9 and 34.5 ng/ml vs 5.1 ng/ml, p<0.001 for both). This was not possible with CK as significantly elevated CK levels were mainly present in active IIM (median 16.5 µkat/L) and the medians of pre-active and stable phase patients (4.8 µkat/L vs 1.7 µkat/L, p=0.060) remained close to normal reference ranges. The area under the receiver operator characteristic curve was 0.87, 0.84, and 0.87 for skTnI and CK individually and combined, respectively.

Conclusion: With the developed skTnI assay, IIM patients were identified from healthy individuals and from patients with traumatic muscular injuries. Also, skTnI was shown to outperform CK in detecting IIM patients in different disease activity statuses.

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