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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