AB1208  FREQUENCY OF MUSCULOSKELETAL AND OTHER EXTRA-INTESTINAL SYMPTOMS IN PATIENTS WITH CELIAC DISEASE

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Background: Celiac disease (CD) is one of the most common gastrointestinal tract diseases, in adults and children. Prevalence of CD is 1%-3% [2]. The most common symptoms of CD are gastrointestinal symptoms. At the same time, celiac disease may manifest with extraintestinal symptoms, including the musculoskeletal, nervous, reproductive system, and skin, especially when it debuts at a late age [1,2]. However, data about musculoskeletal manifestations of CD are limited.

Objectives: To show the frequency of musculoskeletal complaints and their peculiarities in patients with CD.

Methods: Data from 94 patients with diagnosed by gastroenterologist celiac disease were collected with the on-lain survey. All the patients were positive in CD-related immunological and genetic tests and had biopsy established CD.

Results: The average age of respondents is 37.52 ± 11.2 years, women 79 (84,1%), men 15 (15.9%). Among 94 respondents 0.1% do not follow a gluten-free diet, 10.6% <1 year, 25.5% from 1 to 3 years, 11.7% – 4-5 years, 28.7% are on a gluten-free diet >5 years, 14.9% – 10-15 years, 8.5% > 15 years. Gastrointestinal symptoms have started at the age < 10 years old in 59.6% of patients, in 4.2% in 11-16 years old, 13.8% at the age 17-25 years old, 12.8% at the age 26-40 years old, 9.6% had late CD onset (>40 years old). Extraintestinal symptoms such as drowsiness were noted by 46.8%, headaches by 40.4%, weakness by 59.6%, irritability by 57.4% of respondents. Lack of coordination was noticed in 18.1% of cases, dizziness in 22.3%, 57.4% have numbness, decreased sensitivity, and tingling feeling in the limbs. Joint pain had 54.3% of the patients with CD (Figure 1).

Conclusion: Started by 35.1% of patients, without any improvement in 66% of cases. 39.4% of respondents. Non-steroidal anti-inflammatory drugs (NSAIDs) were in 27.6% of patients, and 26.6% was noticed signs of enthesitis. Weakness of arms was noticed by 40.4%, numbness, decreased sensitivity, and tingling feeling in the limbs. Joint pain had 54.3% of the patients with CD (Figure 1).

Figure 1. Frequency of pain in different joints

The maximal intensity of pain was noticed in the morning (8.5%) or late night (13.8%) times and fulfilled inflammatory pain criteria (ASAS). In 17% was noticed interrelation between gluten-free diet violation and the appearance of joint pain. In 28.6% was noticed signs of enthesitis. Weakness of arms was noticed by 39.4% of respondents. Non-steroidal anti-inflammatory drugs (NSAIDs) were started by 35.1% of patients, without any improvement in 66% of cases.

REFERENCES:

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AB1209  MECHANISTIC FACTORS CONTRIBUTING TO PAIN AND FATIGUE IN FIBROMYALGIA AND ME/CFS: AUTONOMIC AND INFLAMMATORY INSIGHTS FROM AN EXPERIMENTAL MEDICINE STUDY

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Background: Fibromyalgia and ME/CFS are multifaceted conditions with overlapping symptoms(1); the pathoetiologic mechanisms are complex and debated(2), however there is a strong association with features of hereditary disorders of connective tissue (hypermobility) and autonomic and inflammatory abnormalities (1,2).

Objectives: To determine potential autonomic and inflammatory mechanisms of pain and fatigue in fibromyalgia and ME/CFS.

Methods: After excluding participants with WCC higher than 10 (suggesting acute infection) baseline markers of inflammation (CRP and ESR) were available for 60 patients with confirmed diagnoses of Fibromyalgia and/ or ME/CFS and 23 matched controls. Participants then underwent full research diagnostic evaluation including a hypermobility assessment(1) and autonomic challenge (60 degree head up tilt; ISRCTN78820481). Subjective pain and fatigue were assessed before and after challenge (VAS). Linear regression models were used to explore predictors, with adjustment for confounders as appropriate. Mediation analyses (looking for mechanistic effects) were conducted according to the method of Hayes (3) and mediation considered significant if bootstrapped confidence intervals of the estimated indirect effect did not cross zero. In these mediation analyses predictor variable was group membership (patient or control), outcome variable was change in 1)pain and 2)fatigue induced by challenge and mediators 1)no of connective tissue features in hypermobility diagnostic criteria endorsed by participant; 2)baseline inflammatory markers.

Results: ESR and CRP were significantly higher in patients rather than controls, even after correcting for BMI, age and sex (B=5.15, t=2.05, p=0.044; B=1.77, t=2.15, p=0.044 respectively). Adjusted ESR and CRP correlated with both subjective fatigue (B=0.44, t=2.09, p=0.04; B=1.63, t=2.60, p=0.011) and pain severity (B=0.13, t=2.51, p=0.014; B=0.45, t=3.01, p=0.004) at baseline. Autonomic challenge amplified pain (B=14.20, t=2.87, p=0.005) and fatigue (B=31.48, t=5.95, p<0.001) in patients to a significantly greater degree than controls, controlling for baseline levels. Baseline ESR and CRP also predicted subjective fatigue was increased by the autonomic challenge (Bootstraped 95% CI of indirect effect do not cross zero, 0.7541 – 7.3888). ESR mediated the degree to which subjective pain was increased by the autonomic challenge (Bootstraped 95% CI of indirect effect do not cross zero,0.1572 – 6.8171). ESR mediated the degree to which subjective fatigue was increased by the autonomic challenge (Bootstraped 95% CI of indirect effect do not cross zero,0.1572 – 6.8171). ESR mediated the degree to which subjective fatigue was increased by the autonomic challenge (Bootstraped 95% CI of indirect effect do not cross zero,0.7541 – 7.3888).

Conclusion: To our knowledge this is the first study to directly explore autonomic and inflammatory mechanisms of pain and fatigue in a combined population of Fibromyalgia and ME/CFS. This study adds to the evidence-base of baseline inflammatory abnormalities in fibromyalgia and ME/CFS. It highlights their potential role in predicting symptom severity and their potential mechanical role in autonomic induced pain and fatigue, suggesting future treatment strategies.

REFERENCES:

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