GOUT AND THE RISK OF PARKINSON’S DISEASE IN THE ELDERLY

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Background: A recent systematic review and meta-analysis, based on two cohort studies and three case-control studies, reported a pooled ratio risk of subsequent Parkinson’s disease (PD) in patients with gout was 0.93 (95% CI, 0.79 to 1.09), a non-significant result. However, statistical heterogeneity was high at 87%-96%, indicating that studies differed from each other. Thus, it is not clear that gout is associated with PD and if so, what is the direction and magnitude of the risk of PD with gout.

Objectives: To assess the association of gout with the risk of incident Parkinson’s disease.

Methods: We used the 5% Medicare sample from 2006–2012 to assess whether a diagnosis of gout was associated with the risk of incident Parkinson’s disease in the elderly. Multivariable Cox regression model adjusted for demographics, Charlon-Roman comorbidity index, common medications, allopurinol and febuxostat use, was used to obtain hazard ratios (HR) and 95% confidence interval (CI).

Results: The mean cohort age was 73 years (standard deviation [SD], 6.5), mean Charlon-Roman comorbidity index score was 1.2 (SD, 1.9), 58% were female, 86% were White and 30% had Charlson-Roman comorbidity index score of >2 (n=15,675). The crude incidence rates of risk PD of 3.3 vs. 1.7 per 1000 person-years in those with gout vs. without gout. Gout was associated with a higher risk of PD in the main analysis, 1.18 (95% CI, 1.10, 1.27). Older age, male gender, White race, higher Charlson-Roman comorbidity index score were associated with higher risk of PD. Sensitivity analyses confirmed main findings. No gender or race differences were noted, but the risk differed by the age: ages 65–75, 75–85 and >85 years were associated with hazard ratios of incident PD with gout of 1.27, 1.12 and 0.98, respectively.

Conclusions: Gout was associated with a higher risk of incident PD in the elderly. The risk of PD with gout was highest in the age group 65–75 years. Mechanisms of this increased risk need to be evaluated in future studies.

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THU0695 DOES HORMONE REPLACEMENT THERAPY PREVENT UNDIFFERENTIATED ARTHRITIS PROGRESSING TO RHEUMATOID ARTHRITIS

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Background: Oral contraceptive (OC) and hormone replacement therapy (HRT) have been reported to have a protective and preventive effect on the progression of rheumatoid arthritis (RA). Although these observations are controversial, progression of undifferentiated arthritis (UA) to RA in pre- and post-menopausal women is largely unreported.

Objectives: To observe patients with undifferentiated arthritis (UA) who were referred to rheumatologists and did not fulfill classification or diagnostic criteria for RA or other connective tissue disease. We studied the efficacy of hormone replacement therapy (HRT) in this setting. In this study, the primary objective was to determine whether HRT reduces joint pain and/or decreases the progression of UA to RA.

Methods: From 2007 to 2016, 1076 patients (male=60, female=1016) classified as UA were referred to one of two clinics because of complaints of joint pains and were enrolled in this study. Beginning in 2012, premenstrual, perimenstrual and postmenstrual women with UA were prescribed ultra-low dose tocopherol (600 mg/day) and HRT. A reduction of over 70% joint pain on a p-visual analogue scale (p-VAS) was set as the criterion of a favourable outcome. Each patient was assigned into one primary disease category. For example, primary SJS was regarded as a disease category but if a patient had secondary SJ, they were assigned to the primary (RA, SLE, SSC) disease category.

Results: During the 5 year observation period, 213/343 (62.1%) had postmenopausal arthralgia (PoMA), 46/112 (41.1%) with premenopausal arthralgia (PoPA), 17/25 (68%) with premenopausal arthralgia (PoMA). In the RA patients, 10.2%, had RF alone, 73.1% (250/342) had ACPA and/Rf or ACPA alone and 16.7% had neither ACPA or RF. The specificity of ACPA was 93.2%. Regarding efficacy of HRT, the incidence of RA in RF positive individuals was 9.1% (5/55) in patients undergoing HRT (current and past user), which was significantly lower (p<0.01) than the 48.4% (30/62) in those never treated with HRT. Likely due to low numbers in the cohort, the incidence of RA in ACPA positive females was 22.2% (2/9) in those receiving HRT was not statistically significantly lower than the 70% (7/10) in those without HRT.

Figure 1 Postmenopausal women responded to conventional HRT in 2018-2015.
Conclusions: The progression of UA to RA is apparently ameliorated in RF positive females who received conventional HRT and oral E3 treatment. Although the numbers were smaller, a significant protective effect was not observed in ACPA positive UA females, because they developed RA before menopause. Our observations suggest that HRT in peri- and post-menopausal and oestrogen (E3) in pre-menopausal females with RF and ACPA positive UA may be important in ameliorating the progression of UA to RA.

REFERENCES:

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Disclosure of Interest: None declared

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THU0697
IS AUTOIMMUNITY RELATED TO NAILFOLD VIDEOCAPILLAROSCOPY PATTERNS PROGRESSION? DATA FROM A TERTIARY CENTRE


Background: Nailfold videocapillaroscopy (NVC) is a non-invasive technique that allows to evaluate the structure and distribution of capillaries in the nail microcirculation.

Objectives: Our objective was to investigate the relation between the autoantibodies (Ab) detected in the patients who undergo follow-up NVCs and the progression from lower to higher severity of the NVCs patterns.

Methods: Longitudinal, observational and descriptive study that includes patients with at least two NVCs, between June 2012 and December 2016 in the Rheumatology service of a tertiary centre. We collected demographics data, number of NVCs performed, Ab positivity, as well as the NVCs patterns. The relationship between postmenopausal hormone therapy on rheumatoid arthritis: the women’s health initiative randomized controlled trials. Arthritis Rheum 2008;59(3):302–10.

Results: Table 1 shows the distribution of the Ab registered in patients before the first NVC, 27 patients did not present positivity to any Ab (23.47%), 28 had isolated ANA + (24.34%), 3 Anticentromere+with or without ANA (28.69%), 7 patients Anti-scl70 + with or without ANA (6.08%), 10 patients Anti-Ro or Anti-La with or without ANA (8.69) and 10 patients presented other types of antibodies than those mentioned (11.3%). The most frequent pattern in the first NVC was non-specific mild alterations (49 cases) in 42.6%, followed by normal pattern in 17.39%, early-scleroderma in 16.52%, non-specific moderate lesions and pattern of late scleroderma. There was a progression from lower to higher severity in 25 NVCs, 88 maintained a similar pattern and 2 NVCs presented significant regression (table 2).

Abstract THU0696 – Table 1. Baseline diagnosis before first NVC.

<table>
<thead>
<tr>
<th>n:25</th>
<th>Autoantibodies</th>
<th>Progression (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 (8%)</td>
<td>Ninguno</td>
<td>1/2: NMI to ES, 1/2: NML to LS</td>
</tr>
<tr>
<td>4 (16%)</td>
<td>ANA+</td>
<td>2/4: NMI to AS, 1/4: NML to ES</td>
</tr>
<tr>
<td>10 (40%)</td>
<td>Anticentromer +</td>
<td>3/10: AS to LS, 3/10: ES to LS, 2/10: NML to ES, 1/10: NMI to ES, 1/10: NML to AS</td>
</tr>
<tr>
<td>3 (12%)</td>
<td>ANA+AntiSc70 +</td>
<td>1/3: NML to AS, 1/3: AS to LS, 1/3: ES to AS</td>
</tr>
<tr>
<td>3 (12%)</td>
<td>ANA+ Anti-Ro/</td>
<td>2/3: NML to ES, 1/3: ES to LS</td>
</tr>
<tr>
<td>3 (12%)</td>
<td>Anti-La+</td>
<td>2/3: NML to ES, 1/3: ES to LS</td>
</tr>
<tr>
<td>3 (12%)</td>
<td>Otros</td>
<td>2/3: NML to ES, 1/3: NMI to LS</td>
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THU0697
RISK OF MAJOR CONGENITAL MALFORMATIONS ASSOCIATED WITH EXPOSURE TO CONVENTIONAL SYNTHETIC DISEASE MODIFYING ANTIRHEUMATIC DRUGS IN WOMEN WITH INFLAMMATORY ARTHRITIS: A POPULATION-BASED COHORT STUDY

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Background: Prior studies of perinatal exposure to conventional synthetic disease modifying antirheumatic drugs (csDMARDs) and risk of major congenital malformations (MCM) have often lacked comparator groups and specific timing of medication exposure.

Objectives: To evaluate the association between csDMARD use before and during pregnancy and risk of MCM.

Methods: We conducted a population-based, retrospective cohort study using British Columbia administrative data from 01/01/2002 and 12/31/2012 on all physician visits, hospital admissions, and dispensed medications, linked to a perinatal registry with valid information on date of conception. We created a pregnancy cohort of women with inflammatory arthritis (IA) using a case definition of 2 ICD9 codes >2 months and ≤2 years apart for rheumatoid arthritis, systemic autoimmune rheumatic diseases, ankylosing spondylitis, juvenile idiopathic arthritis, and psoriatic arthritis. We categorised csDMARDs according to accepted safety research.

Results: None declared