THU0693
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 risk ratio 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 if incident Parkinson’s disease in the elderly. Multivariable Cox regression model adjusted for demographics, Charlson-Roman comorbidity index, common medications, all-cause mortality and hospitalization 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 Charlson-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 incident 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 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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THE EXCESS RISK OF DISABILITY IN PEOPLE WITH NEUROPATHIC PAIN WHEN COMPARED TO THOSE WITH NOCICEPTIVE PAIN IS CAUSED BY PAIN CATASTROPHISING AND PHYSICAL INACTIVITY
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Background: Neuropathic pain (NP) is associated with worse patient outcomes including poorer quality of life and increased disability and mortality when compared to persons with pain that is predominantly nociceptive (NP). It is not known if the higher rate of co-morbid psychosocial factors (e.g. depression, fatigue) in persons with NP explains the increased risk of poor outcomes.

Objectives: To test the hypotheses that pain predominantly of NP origin would be associated with higher levels of disability when compared to NCP, and the excess risk would be independent of putative confounders.

Methods: 1587 participants in a population based prospective study completed a baseline and follow up questionnaire 12 months later. Participants were asked about the presence, location and duration of musculoskeletal pain and they completed the Douleur Neuropathique 4 (scores: 0 indicating no pain, >3 indicating NP). Participants were classified according to their pain reports as having no pain (NP), some pain with (SPn) and without (SP) NP, and chronic widespread pain American College of Rheumatology 1990 criteria) with (CWPn) and without (CWP) NP. The primary outcome was the Stanford Health Assessment Questionnaire (HAQ) from which the Standard Disability Index (HAQ-DI) was calculated. Participants also completed the Hospital Anxiety and Depression Scale (HADS), Chalder Fatigue scale (CF); the Pain Catastrophising Scale (PCS); Rapid Assessment of Physical Activity (RAPA); Social Support scale (SS); Joint Hypermobility scale (JH); Jenkins Sleep Scale (JSS). Ordered logistic regression tested the relationship between pain status at baseline and HAQ-DI at follow up with results expressed as odds ratios (OR) with 95% confidence intervals (CI).

Results: 1235 (77%) participants provided complete data and formed the cohort for analysis. At 12 month follow up the mean (standard deviation) HAQ-DI score was 0.31 (0.62), with higher scores in women and older participants. After adjusting for age and sex, when compared to those with NP at baseline participants with SP were 3 times (OR=2.9, 95% CI (2.2, 3.9)) and those with SPn almost 9 times (8.7 (5.4, 14.0)) more likely to have higher HAQ-DI scores at follow up. Those with CWP were 8 (7.8 (5.7, 10.8)) and those with CWPn 38 (38.1 (23.3, 62.5)) times more likely to have higher HAQ-DI scores. When these associations were adjusted for putative confounders and baseline HAQ-DI scores having pain remained associated with increased HAQ-DI scores at follow up although the relationships were significantly attenuated, and the 95% CIs were similar across pain groups: SP 1.8 (1.3, 2.5); SPn 2.6 (1.4, 4.8); CWP 2.3 (1.6, 3.4); 3.3 (1.9, 6.0). The PCS helplessness scale (1.07 (1.01, 1.13)) and RAPA (0.93 (0.87, 0.99)) were significantly associated with 12 month HAQ-DI scores.

Conclusions: The increased risk of disability in persons with NP was not independent of common pain co-morbidities. Screening and targeting treatment for pain-related helplessness and physical inactivity has the potential to significantly improve disability outcomes for persons with NP.

Disclosure of Interest: None declared

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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 evaluate 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 by simple randomization to one primary disease category. For example, primary SjS was regarded as a disease category but if a patient had secondary SjS, 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 (PeMA), 17/25 (68%) with premenopausal arthralgia (PMPA). In the RA patients, 10.2%, had RF alone, 73.1% (250/342) had ACPA and/or 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 (<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 2013--2015...