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CURRENT ABSTRACTS

Association of kidney disease with prevalent gout in the United States in 1988–1994 and 2007–2010 . . . *By Stephen P. Juraschek, Lara C. Kovell, Edgar R. Miller III, and Allan C. Gelber*

Objective: To determine the prevalence of gout associated with progressive degrees of kidney disease in the US population.
Methods: We performed a cross-sectional analysis among non-institutionalized adults (age 20 and older) of the National Health and Nutrition Examination Surveys in 1988–1994 and 2007–2010. Gout status was ascertained by self-report of physician-diagnosed gout. Chronic kidney disease (CKD) was defined in stages based on estimated glomerular filtration rate (GFR) and single albuminuria measurements (albumin-to-creatinine ratio). Prevalence ratios comparing successive categories of GFR, albuminuria, and CKD as well as temporal trends over a 22-year interval were determined via Poisson regression.
Results: In the US, the crude prevalence of gout was 2–3% among participants without CKD, 4% among participants with CKD stage 1, 6–10% for stage 2, 11–13% for stage 3, and over 30% for stage 4. The adjusted prevalence ratio comparing the CKD stage 4 stratum to participants without CKD was 3.20 (95% CI: 1.96, 5.24) in 2007–2010 and remained significant even after adjustment for serum uric acid. Notably, there was a statistically significant, progressively greater adjusted prevalence ratio of gout associated with successively lower categories of GFR and higher categories of albuminuria.
Conclusions: Among US adults, there exists a strong dose–response association between impaired renal function and prevalent gout. Health providers should be aware of the elevated burden of gout among patients with CKD especially when evaluating new onset joint pain and swelling.
Semin Arthritis Rheum 42:551–561.

Cardiovascular thromboembolic events associated with febuxostat: Investigation of cases from the FDA adverse event reporting system database . . . *By Pranav K. Gandhi, William M. Gentry, and Michael B. Bottorff*

Objective: Uloric (Febuxostat) has been linked with cardiovascular thromboembolic events in gout patients. However, no post-marketing data analysis has investigated these drug-associated adverse event reports. The study objective was to identify febuxostat-associated cardiovascular thromboembolic event reports in the US using the Food and Drug Administration adverse event reporting system (AERS) database.
Methods: Reports listing uloric and febuxostat as the suspect drug and cardiovascular thromboembolic events (combined in a single term based on adverse event reports of myocardial infarction, stroke, among others) as the adverse event were extracted from the drug's approval date through the fourth quarter of 2011. Bayesian statistics within the neural network architecture was implemented to identify potential signals of febuxostat-associated cardiovascular thromboembolic events. A potential signal for the drug-adverse event combination reports is generated when the lower limit of the 95% two-sided confidence interval of the information component (IC), denoted by IC_{025} is greater than zero.
Results: Twenty-one combination reports of febuxostat-associated cardiovascular thromboembolic events were identified in gout patients in the US. The mean age of combination cases was 64 years. Potential signals ($IC_{025} = 4.09$) was generated for combination reports of febuxostat-associated cardiovascular thromboembolic events.
Conclusion: AERS indicated potential signals of febuxostat-associated cardiovascular thromboembolic events. AERS is not capable of establishing the causal link and detecting the true frequency of an adverse event associated with a drug. The positive IC value found in this study merits continued surveillance and assessment of cardiovascular thromboembolic events associated with Febuxostat.
Semin Arthritis Rheum 42:562–566.

Anti-glomerular basement membrane antibody disease treated with rituximab: A case based review . . . *By Uzma A. Syeda, Nora G. Singer, and Marina Magrey*

Objectives: To report the successful use of rituximab in a patient with anti-glomerular basement membrane (GBM) antibody disease and to review the literature regarding rituximab use in anti-GBM mediated disease.
Methods: We report a case of anti-GBM antibody disease with both anti-GBM antibodies and anti-myeloperoxidase (MPO) specific p-ANCA, who developed thrombotic thrombocytopenic purpura (TTP) on high dose prednisone, plasmapheresis, and cyclophosphamide therapy. The patient was then treated with rituximab. We analyzed the clinical features of five additional patients of anti-GBM disease treated with rituximab identified through a systematic literature review.
Results: Our patient was 68-year-old female who presented with acute renal failure. Renal biopsy showed crescentic glomerulonephritis with linear deposits of IgG antibody along the glomerular basement membrane. Treatment was initiated with high dose prednisone, plasmapheresis and oral cyclophosphamide, with subsequent development of leukopenia and TTP and discontinuance of cyclophosphamide. Treatment with rituximab was initiated with clinical improvement of her hematological parameters but not her renal function. Among the five previously reported cases of anti-GBM disease treated with rituximab, three received brief course of IV cyclophosphamide prior to use of rituximab. Except one patient, all recovered renal function and remained dialysis independent. The anti-GBM antibody level remained undetected in all patients.
Conclusions: Combination of prednisone, plasmapheresis, and rituximab can be an effective therapy in patients with an anti-GBM antibody disease complicated with TTP.
Semin Arthritis Rheum 42:567–572.

The association between meniscal damage of the posterior horns and localized posterior synovitis detected on T1-weighted contrast-enhanced MRI—the MOST study . . . *By Frank W. Roemer, MD, David T. Felson, MD, MPH, Tianzhong Yang, MS, Jingbo Niu, MD, Michel D. Crema, MD, Martin Englund, MD, MPH, Michael C. Nevitt, PhD, Yuqing Zhang, DSc, John A. Lynch, PhD, George Y. El Khoury, MD, James Torner, PhD, Cora E. Lewis, MD, and Ali Guermazi, MD, PhD*

Objective: Synovitis is thought to be a secondary phenomenon in the osteoarthritis (OA) process and the menisci might be triggers of localized synovitis. The aim was to assess the cross-sectional associations of posterior horn meniscal damage with perimeniscal synovitis, and with synovitis posterior to the posterior cruciate ligament (PCL) using contrast enhanced (CE) MRI.
Design: The Multicenter Osteoarthritis (MOST) Study is a longitudinal observational study of subjects with or at risk for knee OA. Subjects are a subset of MOST who were examined with 1.5 T CE MRI and had semiquantitative synovitis (scored from 0–2 at 11 locations) and meniscal readings (scored with WORMS from 0–4) available. Logistic regression was used to assess the association of posterior meniscal damage and perimeniscal synovitis in the same compartment, and between posterior meniscal damage and synovitis posterior to the PCL.
Results: Three hundred and seventy seven knees were included (mean age 61.1 years±6.9, mean BMI 29.6±4.9, 44.3% women). The odds for ipsi-compartmental perimeniscal synovitis were increased for knees with medial posterior horn meniscal damage (adjusted odds ratio [aOR] 2.5, 95% confidence intervals [95% CI] 1.3,4.8), but not for lateral damage (aOR 1.7, 95% CI 0.4,6.6). No positive associations were found for meniscal damage and presence of synovitis posterior to the PCL (aOR 0.9, 95% CI 0.6,1.5).
Conclusions: Meniscal damage of the posterior horns is associated with ipsi-compartmental perimensical synovitis. No associations were found for posterior horn meniscal damage with synovitis posterior to the PCL, which suggests that synovitis posterior to the PCL is likely to be triggered by different pathomechanisms.
Semin Arthritis Rheum 42:573–581.

A descriptive analysis of 14 cases of progressive-psuedorheumatoid-arthropathy of childhood from south India: Review of literature in comparison with Juvenile Idiopathic Arthritis . . . *By Alka V. Ekbote, MD, Debashish Danda, MD, DM, FRCP, Sathish Kumar, MD, Sumita Danda, MD, DM, Vrisha Madhuri, MS, MCH, and Sridhar Gibikote, MD*

Background: Progressive-psuedorheumatoid-arthropathy of childhood (PPAC) is an autosomal recessive single gene skeletal dysplasia involving joints. The gene attributed to its cause is WNT1-inducible-signaling pathway protein3 (*WISP3*).
Objective: To study the clinical and radiographic presentation of PPAC in Indian patients and to compare with described features of PPAC and Juvenile Idiopathic Arthritis (JIA) from published literature.
Methods: All cases (n = 14) of PPAC seen in the Rheumatology and Clinical Genetics outpatient clinic between 2008 and 2011 with classical, clinical, and radiological features were studied. The demographic and clinical data were obtained from medical records of the outpatient visits.
Results: Slight female preponderance (57%) and history of consanguinity in parents (43%) was observed in this group. The median age at onset was 4.5 years (range from birth to 9 years of age). Early presentation below the age of 3 years was seen in 3/14 patients (21%) in this group. The growth of all the patients fell below the 3rd percentile for the age. Historically, hip joint involvement was the most common presenting feature; however, elbow, wrist, knees, feet, spine, shoulder joints and small joints, namely proximal