



SPAR Reporter

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A Case of Treatment Failure Gout? Maybe. Maybe not.

Submitted by Joan McTigue, PA-C

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Treatment failure gout can refer to failure to achieve the recognized target serum urate level of 6.0mgs; failure to suppress or stop flares; or failure to prevent the progression of disease. Unfortunately, it is not uncommon for all three to transpire in one case.

The causes of so-called *treatment failure gout* fall on the shoulders of both patients and medical professionals caring for them. For patients, non-adherence and poor understanding of their disease can couple with poor communication with providers. For the providers, medical professionals often work with a limited number of therapeutic options, especially when co-morbidities are strongly featured. Or, there can be fundamental lack of understanding of the goals for urate-lowering therapy and pain control. Sadly, providers can simply give up or feel their strategy is 'good enough' when they should refer the patient on to those more skilled with complex cases of hyperuricemia and gout.

The following case embodies all of the previously mentioned issues.

Mr. H was a 40-year old white man with a 15 year history of gout which presented with recurrent episodes of very painful, hot lower extremity joint effusions that lasted 4-7 days. He was initially just seen in emergency rooms. He would try to self-medicate himself through the flares with OTC products typically visiting the Emergency Room many days into the flare. He was finally referred to our sub-

specialty group a week after an especially bad flare. Mr. H had come in 4 times that year.

This first visit occurred 7 years ago. His joints were cool the day we saw him and he had no tophi. His serum acid was 10.0mgs/dL. He history with his gout was so classic that we used the visit to educate him about gout as a chronic condition. We started him on allopurinol 100mgs titrating him to 300mgs along with daily colchicine 0.6mgs BID. After 3 visits he did not return and he only saw his PC team sporadically in the interval years. At some point, his allopurinol prescription expired and his uric acid was left unchecked. He was in stage two gout—the inter-critical phase which is common for both patients and provider to keep the gout off the clinical radar.

We received a request to see Mr. H last summer, this time from the PC group. They said he was a "man with gout who failed allopurinol therapy." At that time, his gout flares were came monthly, lasted a full week, and his job as a truck driver was becoming compromised.

At that time, he was on Prednisone 10mgs and two weeks had passed from the last flare. He was going through a relatively quick steroid taper. Unfortunately, the most recent flare had not ended. His knee was warm with a modest effusion and mild pain.

We drained the knee of 30mgs of synovial fluid which was culture negative, MSU crystal positive .

At the time of the arthrocentesis, we instilled 40mgs of Kenalog into the knee. We slowed down the rate of his steroid taper and prescribed daily colchicines, 0.6mgs BID. It was imperative to give him an authentic, lasting relief and to break the flare to initiate urate lowering therapy. His uric acid was 10.2mgs. It is typical to wait for the flare to be fully subsided before creating urate pool perturbations which initiating urate lowering therapy will bring on.

While Mr. H was calm enough to hear certain messages about his gout treatment goals, he was pre-occupied by fear of another flare. He was not very confident he could cut back on beer consumption as he regularly consumed a 6-pack each day during weekends. Though hesitant, he agreed to stop his weekend beer drinking for a month.

He complied with most of the program and, at his next visit 3 weeks later, was free of pain and swelling and was down to 10mgs of Prednisone. However, he had stopped taking the colchicine fearful it would give him diarrhea as it had in the past during previous ER visits where he was placed on a high dose.

He was on amlodipine for mild HTN, so we switched him to Losartan for its mild, but real uricosuric effect. We knew we had to overcome his bad experiences with high dose colchicine before he would let us put him back on colchicines BID. We explained the dosing differences and its current role as flare prophylaxis in contrast to how it had been used previ-

A Case of Treatment Failure Gout? Maybe. Maybe not.

Submitted by Joan McTigue, PA-C (Continued)

ously. We also initiated allopurinol at 100mgs per day.

One month later, he was down to Prednisone 7.5mgs, was still tolerating colchicine BID, and his uric acid was 8.0mgs on 100mgs of allopurinol. We drained his right elbow which had a large, painful, warm olecranon bursa of 15cc of MSU-laden synovial fluid; and instilled 10 mgs of depomedrol into the bursa. We stressed ice and rest for the joint.

We kept him on Prednisone at 7.5mgs, colchicine BID and began Anakinra 100mgs weekly given by a self-administered SCut injection. We stopped allopurinol as it was proving diffi-

cult to titrate and started febuxostat at 40mgs and added Vitamin C 500mgs for its mild uricosuric affect.

He called our office flare-free in three weeks. We advised him to continue to taper the prednisone, but to continue the Anakinra weekly injections. Five weeks later, he was flare-free and his uric acid was 6.2mgs/dL. We kept him on colchicine and febuxostat, and stopped use of Anakinra. We increased his vitamin C to 1,000mgs a day and stressed the role of triggers like beer binging.

We saw him 3 months later and he was no longer drinking at all. He was on the same regimen

and was flare-free with a uric acid of 6.0mgs. He was happy and we were happy.

We plan to see him at 4 month internals for monitoring. We stressed the role of flare-triggers like beer, the need for lifelong urate lowering medication, and confirmed our plan to stop colchicine once his uric acid stays at, or below, 6.0mg/dL for several months and he remains flare-free. If his flares returns, we begin a Medrol dose pak immediately.

Though this case posed many challenges, we were able to use all of the tricks (and drugs) in our bag to get Mr. H to target and pain-free.

ACR Annual Scientific Meeting

Physician Assistants in rheumatology who attended the 2010 American College of Rheumatology Scientific Meeting enjoyed the presentations, exhibits, and networking opportunities. Attendees shown at the conference in Atlanta, Georgia include SPAR President Don Flinn, PA-C; SPAR Secretary Susan Richmond, PA-C; SPAR Director-at-Large Barbara Slusher, PA-C; SPAR Member Ben Smith, PA-C; and Neil Moody, PA-C.



News You Can Use

Pam Moyers Scott, PA-C, MPAS, DFAPPA

Title: [Proton pump inhibitor use, hip fracture, and change in bone mineral density in postmenopausal women.](#) (*Arch Intern Med* 2010; 170:765-771)

Summary: This article reports the findings of a retrospective chart review conducted on the 161,806 women enrolled in the Women's Health Initiative of which 3,396 women were identified as being on proton pump inhibitors (PPIs). A multivariate analysis revealed that women on chronic PPIs had nearly a 50% increased incidence of vertebral fractures and a 25% increase in wrist fractures. However, there was no difference in the incidence of hip fracture between chronic PPIs users and non-users. Interestingly, they only had a modest effect on bone mineral density. Additionally, the reviewers found that the 10,016 women who were identified as being on H₂ receptor antagonist therapy did not have a greater incidence of hip, wrist, or vertebral fractures. Never-the-less, these women experienced an increased incidence overall fractures.

Comments: Previous studies exploring the relationship between PPIs and osteoporotic fractures have met with conflicting results. This could be due, at least in part, to the fact that several of these studies looked at hip fracture incidence only. Unfortunately, this prospective study does not clarify the adequately address the issue. Because osteoporosis and osteoporosis fractures are a complex metabolic condition, there could be other interfering factors that have not been adequately explored to date. For example, perhaps PPIs marked gastric acid suppression interferes with the adsorption of calcium; however, it might be minimized by an adequate vitamin D intake or acquiring the mineral from dietary sources instead of supplements. Thus, it is evident that there are many more factors that must be carefully evalu-

ated before a conclusion can be reached. Ideally, this investigation should take the form of a large, long-term, prospective clinical trial.

In the interim, physician assistants (regardless of practice setting) should continue to exert sound medical judgment and only prescribe medication in which the benefits appear to clearly outweigh the risks. Currently, this includes continuing PPI therapy in the patient's who are taking long-term NSAIDs for their cytoprotective properties. Furthermore, we should inform our patients of these conflicting findings before prescribing (or renewing) these medications so that the most current clinical data can be reviewed, a personal risk-hazards ration can be established, and the patient can provide informed consent. Therefore, until more data is available, PAs, who practice in rheumatology, should not considering ignoring the standard of care of prescribing a PPI for cytoprotection to appropriate patients who are currently taking, or anticipated to be taking, chronic NSAID therapy or other medications associated with peptic ulcer disease and hemorrhage.

Title: [Abnormal over expression of mastocytes in skin biopsies of fibromyalgia patients.](#) (*Clinical Rheumatology* 2010. DOI: 10.1007/s10067-010-1474-7)

Summary: This article describes the findings of formalin-fixed, paraffin-embedded, skin biopsies performed on 63 patients with fibromyalgia syndrome (FMS) and 49 healthy volunteers. The authors discovered that the patients with FMS had a significantly larger concentration of mast cells in their papillary dermis (5 to 14 vs. 0 to 1).

Comments: The exact significance of this unique finding is unknown. Mast cells are stimulated by a variety of physical, emotional, biochemical, and environmental triggers. Their increase is most frequently associated with aller-

gic processes. This is likely to lead to clinical trials exploring treatments traditionally associated with allergic processes including more medications from the antihistamine family (like doxepin) and perhaps even leukotriene inhibitors and corticosteroids. However, until double-blind placebo control trials can be conducted providing evidence based information that these treatments are indeed beneficial, PAs in rheumatology (and other specialties dealing with FMS patients) should use these other agents only when all other options have been exhausted and with informed patient consent has been obtained.

Title: [Clinical optical coherence tomography of early articular cartilage degeneration in patients with degenerative meniscal tears.](#) (*Arthritis & Rheumatism* 2010;622(5):1412-1420)

Summary: This study evaluated 30 consecutive patients who underwent arthroscopy for degenerative meniscal tears, of which only 23 had evidence of a cartilage abnormality on arthroscopy. The authors compared the patients' arthroscopy results to preoperative MRI T2 values and optical coherence tomography (OCT) findings. They concluded that the correlation between both the qualitative and quantitative OCT scores and the arthroscopy results ($P=0.004$) and significantly superior to the correlation between the arthroscopy and MRI results ($P=0.03$)

Comments: OCT is a relatively new technology that utilizes near-real time scanning to produce high-resolution images of the cell layers of biological tissues. Recent advances in the technology have given it the potential to revolutionize the imaging field. Its first successful clinical application was to provide a detailed 3-D image of the retina and related structures. Now, it is also capable of evaluating the anterior portion of the eye. It is being utilized to identify very early changes (frequently before the development of symptoms) associated with glaucoma, macular

News You Can Use

Pam Moyers Scott, PA-C, MPAS, DFAPPA (continued)

edema, macular holes, retinopathies (including diabetic), atrophy of the optic nerve head, and abnormalities of the retinal nerve fiber layer (including optic neuritis).

It is also being evaluated as a non-invasive technique to assess vascular status of brain (and other internal) tumors as an indicator of their malignancy potential. Furthermore, it is being evaluated intraoperatively to assist in limiting the amount of surround tissue that must be removed to obtain tumor-free margins in certain malignancies where preservation of the surrounding tissue is essential (e.g. brain). This study indicates that it is superior to MRI in identifying meniscal tears due to osteoarthritis. However, due to the small study size, much larger studies are going to be required before this becomes the standard of care. Additionally, studies on other joints are going to be essential to ensure these findings can be reproduced at other sites. If they can, it is likely this will become the technique of choice to evaluate not only painful joints but asymptomatic ones in at risk patients in hopes of preventing diseases such as osteoarthritis. Furthermore, it could have a dramatic role in evaluating the response to orthopedic treatments in patients with osteoarthritis and possibly other arthropathies. The OCT requires much less time to perform than an MRI and much less expensive equipment. It could easily be available in orthopedic and rheumatology offices. At this time physician assistants in rheuma-

tology and related fields need to stay abreast of studies conducted on the application and accuracy of this technique. If preliminary results can be reproduced and applied to other joints, PAs should consider adding this to their scope of practice and obtaining training to perform the procedure.

Title: [Omega-3 fatty acids infusions as adjuvant therapy in rheumatoid arthritis](#). (*J Parenter Enteral Nutr* 2010; 34(2):151-155)

Summary: This article outlines the results of a double-blind, randomized, placebo-controlled trial of 23 patients with moderate to severe RA who were randomly assigned to receive IV fish oil emulsion followed by oral fish oil supplements or a placebo infusion and pills. The author found that joint counts for both swollen and tender joints were significantly lower in the treatment group.

Comments: Omega-3 (and other fish oil) oral supplements have been studied in the past as a possible treatment for RA. Although it appears to have some anti-inflammatory properties (based on its benefit on endothelial dysfunction), these preliminary trials met with disappointing results. Thus, the approach the authors took is rational to achieve much higher concentrations of the supplement. Although this study appears to be well constructed and conducted, it still only involved 23 patients of

which approximately half received the actual treatment. Therefore, before a physician assistant in rheumatology recommends this treatment, further large-scale, longer-term trials are required.

Title: [Intracortical remodeling and porosity in the distal radius and post-mortem femurs of women: a cross-sectional study](#). (*Lancet* 2010; 375: 1729-36)

Summary: This cross-sectional study from Australia evaluated the distal radius of 122 women via high-resolution peripheral CT. The authors discovered that over 2/3 rds. of bone loss in women between the ages of 50 and 80 years occurred in cortical, not trabecular bone. Furthermore, their findings revealed that the majority (84%) of this loss occurred after the age of 65 years. Furthermore, remodeling within the area of the cortex immediately adjacent to the bone marrow represented nearly half of all the bone loss. The authors concluded that osteoporosis treatments should be focused on cortical, not trabecular, bone and that traditional DEXA scanning actually underestimates age-related changes by approximately 4-fold because cortical remnants are included in the measurement.

Comments: The findings of this small study certainly contain some food for thought. They are plausible when one considers, with the exception of vertebral fractures, all fractures are cortical in nature. Furthermore, it would assist in explain-

News You Can Use

Pam Moyers Scott, PA-C, MPAS, DFAPPA (continued)

ing why the majority of postmenopausal women diagnosed with osteoporosis never have a non-vertebral fracture. Obviously, these findings are going to need to be reproduced in much large patient populations before they are considered reliable. If found to be accurate, this will have a dramatic impact on the way we diagnose and treat osteopenia and osteoporosis. While awaiting these results, the best treatment physician assistants can offer women for osteoporosis is prevention. This supports the important of ensuring all individuals, regardless of age or gender, have an adequate calcium and vitamin D intake, exercise regularly (weight bearing and strength training) and eliminate other potentially modifiable risk factors (e.g. smoking and excessive alcohol intake).

Title: [Atherosclerosis in early rheumatoid arthritis: very early endothelial activation and rapid progression of intima media thickness.](#) (*Arthritis Res Ther* 2010; DOI: 10.1186/ar3116)

Summary: This small study from Sweden looked at intima-medial thickness via ultrasonography as well as serum levels of soluble intercellular adhesion molecule (sICAM), monocyte chemoattractant protein 1 (MCP-1), Von Willebrand factor (vWF) in 79 patients with RA (diagnosed before the age of 60 years) and 44 age- and gender-matched controls in an attempt to explain why patients with RA experience premature atherosclerosis. Despite therapy with DMARDs and biochemical markers indicating low levels of inflammation, the patients with RA had greater intima-media thicknesses and higher baseline levels of the aforementioned serum studies. These changes were unaffected by other traditional cardiovascular risk factors (e.g. hypertension, hyperlipidemia, age, gender, and smoking status).

Follow up of 27 patients and their controls occurred at 18 months after the initial evaluation. Despite endothelial activation being present, no evidence of endothelium dysfunction was evident. Regression analyses conducted at that time actually revealed an inverse relation-

ship to the patient's disease activity (as measured by DAS28 scores) and soluble (s) L-selectin (an adhesion molecule down-regulated in chronic inflammation) levels. After adjusting for inflammation, multiple linear regression models still revealed a significant relationship between soluble (s) L-selectin levels and endothelial dysfunction.

Comments: This is another small study that is going to require much larger patient populations to confirm (or refute) these findings. This is significant because, if correct, it disproves the current assumption that the premature atherosclerosis in RA patients results from an inflammatory effect of the endothelium. At this time, physician assistants in rheumatology should be vigilant to not only control signs, symptoms, and inflammation due to RA but evaluate for risk factors for cardiovascular disease and aggressive treat any that are present to hopefully prevent premature atherosclerosis and its complications.



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SPAR DAY at the AFPPA 2011 Fall Conference

Join fellow PAs and other clinicians in Monterey, CA at the Portola Hotel November 9-12, 2011, for the 13th Annual Fall CME Conference.

Learn from the best instructors, providing hands-on workshops and interactive core sessions to help you improve existing skills and learn new ones.

SPAR will have a day dedicated to rheumatology. The rheumatology sessions are scheduled on Friday, November 11. Conference goals include presenting comprehensive therapeutic approaches in the field of rheumatology to improve patient outcomes.

The target audience for the SPAR track is physician assistants and nurse practitioners in a clinical rheumatology and/or family practice setting.

SPAR members and AFPPA members receive \$120 off the conference registration. SPAR dues are only \$40 per year, or \$10 for students. To join now go to www.rheumpas.org.



For more information or to register visit www.afppa.org

SPAR AGENDA

Friday, November 11, 2011

8:00 – 9:00 AM

Hand Surgery for the Arthritic Patient

Speaker Don Flinn, PA-C

9:00 – 10:00 AM

Juvenile Idiopathic Arthritis

Speaker Elizabeth Roth-Wojcicki, RN, MS, CPNP

10:00 – 10:15 AM.....**Break**

10:15 – 11:45 AM

Taking the Blindfold Off with Ultrasound-Guided Joint Injections

Speaker Patrick Astourian, PA-C

11:45 AM – 1:00 PM.....**Lunch**

1:00 – 2:00 PM

Osteoporosis Update

Speaker Steven Harris, MD

2:00 – 3:00 PM

Rheumatoid Arthritis & Cardiac Risk Factors

Speaker Dimitrios Pappas, MD

3:00 – 4:00 PM

Systemic Lupus Erythmatous

Speaker David I. Daikh, MD

4:00 – 5:00 PM

Gout & Crystal Arthroscopies

Speaker Joan McTigue, PA-C

SPAR Membership Information

Member Benefits

- SPAR Reporter newsletter (3 issues per year) featuring *Expert Consults*, case studies, personal accounts of PAs in rheumatology practice, and more.
- Rheumatology website access to www.rheumpas.org featuring advanced professional practice information tailored to your needs in rheumatology.
- Relationships with & representation at other professional organizations including AAPA, ACR & ARHP
- Opportunities to teach, volunteer, lead and mentor!

Annual Dues Expire 6-30-11!

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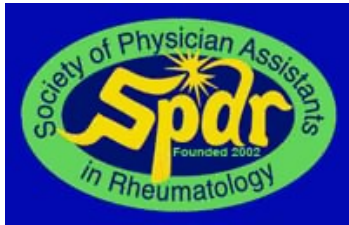


SPAR's CME Chair, Rick Pope, PA-C, with his PA students at Quinnipiac University.

QU PA Students:

Todd Bruss
Sharon Tokarz
Jonathan Shepard
Michael Dziubina
Richard Blanchrd
Claire Fortier
Erica Bertocin
Robin Fuesserich
Felicia Sancez





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Recent Advances in Gout Treatment archived webinar with post-test

Learning Objectives: At the conclusion of this session, the participant will be able to:

- Diagnose and manage patients with gout.
- Describe the role that diet plays in gout.
- Describe appropriate therapy for gout.

Target Audience: This program is designed for physician assistants (PAs), nurse practitioners (NPs), physicians (MDs/FPs) and nurses (RNs/LPNs) who care for patients with gout in a clinical, surgical or research setting. Content will be geared to advanced practice professionals.

Presenters:



Robert Wortmann, MD, MACR
Professor of Medicine
Section of Rheumatology
Dartmouth Medical Center
& Dartmouth Hitchcock Medical Center
Lebanon, NH



Joan McTigue, PA-C
Physician Assistant
Division of Rheumatology
University of Florida Medical Center
& VA Medical Center
Gainesville, FL

Accreditation:

PHYSICIAN ASSISTANTS

This program has been reviewed and is approved for a maximum of one hour of AAPA Category I CME credit by the Physician Assistant Review Panel. Approval is valid for one year from the issue date of May 24, 2011. Participants may submit the self-assessment at any time during that period.

This program was planned in accordance with AAPA's CME Standards for Enduring Material Programs and for Commercial Support of Enduring Material Programs.

NURSES/NURSE PRACTITIONERS

This program has been approved for 1.0 contact hours to be provided by the Florida Board of Nursing Provider #50-7071. Each nurse should claim only those hours that he/she actually attends an educational activity.

Each attendee should claim only those hours that he/she actually attends.



This program was supported by an educational grant from URL Pharma