FROM THE PUBLISHERS OF AUSTRALIAN DOCTOR

Rheumatology update

EDITOR: NICOLA.GARRETT@REEDBUSINESS.COM.AU | ADVERTISING: BRYN.MCGEEVER@REEDBUSINESS.COM.AU 14 NOVEMBER 2011

# Lifestyle intervention halves knee OA pain

Tony James

**ACR 2011, Chicago:** Intensive weight loss combined with low to moderate exercise can halve the pain suffered by overweight patients with knee osteoarthritis, research suggests.

Investigators including Professor David Hunter from Royal North Shore Hospital in Sydney, enrolled more than 450 patients aged at least 55 who had a BMI between 27 and 42 kg/m$^2$ in the international study.

Patients were randomised to either intensive dietary restriction using meal replacement products; two 15-minute walks and one 20-minute weight training session three times weekly; or the combination. The diet aimed for 10% weight loss.

At 18 months, patients lost an average 11kg (11% of original weight) with diet and exercise and 9kg with diet alone, but only 2kg with exercise alone.

**Reductions in pain were apparent at six months**

The WOMAC pain score fell 51%, from 6.7 at baseline to 3.3, in patients who both dieted and exercised. This compared to reductions of 27% and 29% in the other two groups.

Other benefits included improvements in the WOMAC functional score and in walking speed.

Improvements in weight, pain and function were apparent in all groups after six months, but continued to grow in the diet and exercise group until the end of the study.

"Long-term intensive weight loss is possible in an OA population," said study co-author Dr Stephen Messier from Wake Forest University in North Carolina.

"Clinicians can tell their patients they will see marked improvement within six months, and with a combination of diet and exercise the benefits will continue to grow.

"These data provide evidence that the best recommendation for long-term symptom reduction in overweight and obese patients with knee OA is intensive weight loss combined with low to moderate intensity exercise."

**Tony James attended ACR 2011, Chicago, courtesy of Pfizer Australia.**

# Belimumab: the first SLE-specific biological?

Tony James

**ACR 2011, Chicago:** Belimumab, a human monoclonal antibody which inhibits B-lymphocyte stimulator (BLyS) is emerging as an effective and safe treatment for patients with active systemic lupus erythematosus, research suggests.

Dr Joan Merrill, from the Oklahoma Medical Research Foundation, reviewed long-term data from 296 patients who continued on treatment for up to six years after participating in a phase 2 dose-finding trial. Exposure to belimumab totalled about 1500 patient-years.

Rates of adverse events remained stable or reduced throughout the follow-up period.

There were five deaths recorded, but no single cause predominated. They were attributed to aspiration pneumonia with subsequent sepsis and respiratory failure, infection, cardiovascular disease, and suicide.

"Belimumab added to standard SLE therapy was well tolerated for its intended indication in patients remaining on treatment over six years," Dr Merrill said.

Autoantibody-positive patients treated with belimumab showed sustained improvement in disease activity, a decline in the BILAG organ domain score and a reduction in flares during the six years of follow-up, accompanied by reductions in corticosteroid use and autoantibody levels.

Another review of 2,133 patients from a phase 2 study and two phase 3 studies concluded the novel treatment was "generally well tolerated" when used in combination with a wide range of standard SLE therapies.

The findings complemented the results of the pivotal BLISS-52 study published in the *Lancet* in February. The one-year phase 3 trial, including Australian patients, demonstrated a significant reduction in disease activity.

It concluded that belimumab had the potential to be the first targeted biological treatment approved specifically for SLE.

Belimumab has been approved for use in the United States, Canada and Europe, and is under consideration for approval in Australia.

Rheumatology update

FROM THE PUBLISHERS OF AUSTRALIAN DOCTOR

EDITOR: NICOLA.GARRETT@REEDBUSINESS.COM.AU | ADVERTISING: BRYN.MCGEEVER@REEDBUSINESS.COM.AU 14 NOVEMBER 2011

# ACR gout guidelines to include gene testing advice

Tony James

**ACR 2011, Chicago:** Preliminary guidelines from the American College of Rheumatology recognise advances in gout diagnosis and treatment, including recommending genetic screening for allopurinol hypersensitivity.

Professor Robert Terkeltaub, from the VA Medical Center in San Diego, outlined the proposed recommendations for urate-lowering therapy and the treatment of chronic tophaceous gouty arthropathy. The guidelines are yet to be formally ratified after a peer review process.

"Many of the issues will be second-nature to rheumatologists, but there is a need to improve the standard of care in non-specialist practice," he said.

High-resolution ultrasound is now recognised as a valid and useful imaging modality in symptomatic gout, but evidence of urate crystals should not be the sole justification for starting medication, he said.

**Genetic testing should be considered in Han Chinese patients**

An algorithm for urate-lowering medication specifies the indications for starting treatment, including the presence of tophi or two or more attacks of gout a year.

It confirms allopurinol as the first-line choice, with febuxostat (not yet available in Australia) being an alternative xanthine oxidase inhibitor in patients intolerant of allopurinol.

Probenecid is the first choice when a uricosuric agent is needed, and it can be combined with a xanthine oxidase inhibitor.

"Treatment should be titrated to achieve a serum urate level of less than 6 mg/dL, but a reduction to 5 mg/dL might be needed to control signs and symptoms," he said.

Allopurinol should be commenced at no more than 100 mg/day, titrated every 2-5 weeks to a maximum of 300 mg/day.

"For the first time, we suggest that you consider selective pharmacogenetic screening for the HLA-B*5801 allele, which increases the risk of allopurinol hypersensitivity, in high-risk populations including Han Chinese," Professor Terkeltaub said.

Pegloticase, a recombinant uricase that is available in some markets, should be reserved for patients with severe gout who are refractory to, or intolerant of, conventional drugs that have been dosed at appropriate levels, he said.

The guidelines also include recommendations on acute gouty arthritis and anti-inflammatory prophylaxis.

**What do you think?**

**Click here to comment**

Rheumatology update

FROM THE PUBLISHERS OF AUSTRALIAN DOCTOR

EDITOR: NICOLA.GARRETT@REEDBUSINESS.COM.AU | ADVERTISING: BRYN.MCGEEVER@REEDBUSINESS.COM.AU 14 NOVEMBER 2011

# Brushing your teeth might prevent RA

Tony James

**ACR 2011, Chicago:** Better dental hygiene might prevent rheumatoid arthritis and even improve joint symptoms once the disease is already established, experts say.

Dr Michele Ravenal, from the Medical University of South Carolina, said patients with RA were up to eight times more likely to develop significant periodontitis as the general population.

"One theory is that the relationship is... related to common risk factors such as smoking," Dr Ravenal said.

"But evidence is growing that the association is causal, given the surprising similarities between the pathology of periodontitis and the pathology of RA."

At least 20 different bacterial pathogens were known to adhere to the plaque biofilm that accumulated in gingival pockets around the crowns of teeth, she explained.

Chronic inflammation eroded the soft tissue and bone, leading down a pathway of gingival recession, plaque calcification, weakening of the periodontal ligament, pain, and eventually loss of the tooth.

Both RA and periodontitis involved chronic inflammatory reactions

Both RA and periodontitis involved chronic inflammatory reactions in immunogenetically-susceptible patients, with HLA-DRB1 alleles contributing to the risk for both conditions. They shared IL-1 as a primary inflammatory mediator.

Bone destruction – around the jaw in periodontics and in the joint is RA – occurred in a similar way, driven by the same sets of inflammatory cytokines.

And bacteria known to be periodontal pathogens had been identified in the blood and synovial fluid of RA patients.

Preliminary studies had suggested that effective treatment of periodontitis could reduce DAS28 scores and ESR in patients with RA, Dr Ravenal said.





# Clear identity needed for musculoskeletal health

Tony James

**ACR 2011, Chicago:** 'Musculoskeletal health' needs a stronger identity that encompasses the many specialties that help patients with bone, joint and muscle diseases, according to British rheumatologist Professor Tony Woolf.

"For example, 'cancer' has a clear, unified identity to the public, clinicians, politicians and decision-makers, even though it is a varied collection of diseases," he told the ACR meeting.

"The same applies to 'mental health' or 'heart disease'. The focus is on the burden of a group of related diseases, rather than a competition between individual illnesses, and this approach is very effective in lobbying for better resources."

Professor Woolf, from the Royal Cornwall Hospital in the UK, was a key figure in the UN Bone and Joint Decade (2000-2010) and is leading a subsequent advocacy and education program, the Global Alliance for Musculoskeletal Health.

Musculoskeletal diseases were disadvantaged in the race for resources because they had a relatively low mortality risk despite their enormous morbidity, he said.

"These diseases are very common in all countries and all cultures, and have a pervasive, detrimental effect on quality of life, function and productivity. There are many effective treatments, but they are often under-used, or used too late, or are not equitably available."

An acceptance of disability and pain as a normal consequence of ageing, the stoicism of some patients, and a failure of clinicians to implement evidence-based treatments all contributed to the unnecessary costs, he said.

**What do you think?**
**Click here to comment**