

Rheumatology

update

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Cartilage biomarkers identify knee OA subgroups

Laura Macfarlane

Cartilage biomarkers can be used to identify subgroups of patients with knee osteoarthritis (OA) who have different rates of disease progression, an Australian study has found.

"This study is particularly novel as very few studies have examined the relationship between cartilage biomarkers and cartilage volume loss assessed by MRI, as well as a joint replacement, a clinically important outcome," the authors wrote in the *Annals of Rheumatic Diseases*.

The study conducted by researchers from the Monash University Medical School in Victoria assessed around 120

subjects with knee OA over the age of 40 years, who had MRI at baseline and at two years.

They measured serum cartilage biomarkers - cartilage oligomeric matrix protein (COMP), N-propeptide of type IIA procollagen (PIIANP) and collagen type II cleavage (C2C) - at baseline and determined change in knee cartilage volume over two years and knee joint replacement over four years.

The participants were divided into subgroups with high or low cartilage biomarkers and the relationships between outcome measures and biomarkers were examined in the whole population and separately in marker subgroups.



The study data showed that the relationship between cartilage biomarkers was not linear across the whole population. COMP and PIIANP were significantly associated with reduced rate of cartilage loss in the low, but not the high, marker subgroups and C2C was associated with in-

creased lateral cartilage volume.

"Similar results were obtained when the subject receiving bisphosphonate treatment and the subjects receiving glucosamine were excluded from the analysis," the authors noted.

The authors said their data suggested that cartilage biomarkers may be used to identify subgroups within a knee OA population with different disease progression rates.

"This may facilitate our understanding of the pathogenesis of disease and highlights the heterogeneity of knee OA," they concluded.

Annals of Rheumatic Diseases 2010 published online before print.

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Surprising twist in gout-diabetes link

Sarah Colyer

Diabetes may have a protective effect against gout, surprising new research suggests.

Men with diabetes had a 41% lower risk of gout compared with non-diabetic men, a study matching almost 25,000 gout patients with controls found.

The risk of developing gout dropped significantly with each additional year an individual lived with diabetes, from a 19% risk reduction after three years, to a 27% risk reduction after 10 years, the study found, with the largest risk reduction occurring for



Men with diabetes had a 41% reduced risk of gout

patients with longstanding type 1 diabetes or severe type 2 diabetes.

Writing in the *Annals of Rheumatic Diseases*, the authors

acknowledged their findings "might appear counterintuitive" given the strong associations between hyperuricaemia, gout and the metabolic syndrome, which was considered a pre-diabetic state.

However, they said diabetes could protect against gout because of its association with reduced serum uric acid levels and impaired inflammatory response.

"While gout increases the future risk of diabetes, diabetes decreases the future risk of gout," they added.

Rheumatologist Professor Ric Day, of St Vincent's Hospital in Sydney, said the findings

were surprising but plausible.

He said in those diabetic patients who were at risk of gout, the findings suggested lifestyle modifications may be effective.

"We could just relax a little bit with respect to the risk of acute gout. It gives us a bit more latitude before we reach for the pen and prescribe allopurinol in these patients," he said, adding allopurinol should only be prescribed for people with recurrent attacks of gout.

Annals of Rheumatic Diseases 2010; online.

Australian Doctor

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Scope to improve gout treatment

Tony James

Better use of existing treatments could lead to immediate and substantial improvements in the control of gout, according to Sydney rheumatologist Dr Neil McGill.

Writing in the *Journal of Internal Medicine*, Dr McGill **noted** that allopurinol remained the mainstay of therapy, but dosing was often suboptimal and not adjusted to achieve treatment targets.

Guidelines on the target varied, but a plasma urate level <0.36 mmol/L was desirable and <0.30 mmol/L even better. The vast majority of allopurinol prescriptions were for doses of 300 mg/day or less, even though only 24% of patients would be expected to have a urate level <0.30 mmol/L at this dose, Dr McGill said.

It would be almost inconceivable for a doctor to prescribe an antihypertensive and not



Allopurinol dosages are often suboptimal and not adjusted

monitor blood pressure, but it was disturbingly common, and equally inappropriate, to

prescribe urate-lowering treatment and not check whether the target was being met, he said.

Slow dose titration, reducing urate by about 0.04 mmol/L per month, was essential to prevent flares of gout.

Probenecid was an appropriate option for patients with allopurinol hypersensitivity. New treatments including febuxostat and pegloticase would probably become available in Australia, but at considerable cost.

Dr McGill emphasised the necessity of an accurate diagnosis in new cases of gout, based on synovial fluid analysis.


Internal Medicine Journal 2010; published online.

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Denosumab and alendronate have different effects on bone

Michael Slezak

Denosumab and alendronate not only have different mechanisms of action, but have different effects on bone micro-architecture and strength, according to new research.

The double blind placebo controlled trial of 247 postmenopausal women with T-scores between -2.0 and -3.0 found that while the bisphosphonate alendronate slowed bone loss, the fully human antibody denosumab partly restored cortical density.

“The reason for this is being studied, but our provisional data suggest that denosumab reduces cortical porosity by allowing filling or partial filling of porosity already present and by preventing the appearance of newly excavated pores more so than does alendronate,” lead author Dr Ego Seeman told *Nature* journals.

The researchers measured total, cortical, and trabecular BMD



Both drugs have different effects on bone micro-architecture

as well as cortical thickness at the distal radius and the tibia at baseline and after 12 months.

Alendronate prevented the decline of these variables seen in the placebo group, while denosumab improved them.

In addition, the polar moment of inertia increased more in the denosumab group than in the

alendronate or placebo groups.

“These data suggest that structural decay due to bone remodeling and progression of bone fragility may be more effectively prevented with denosumab,” the researchers wrote in the *Journal of Bone and Mineral Research*.

Journal of Bone and Mineral Research 2010; Online.

Family histories neglected: audit

Michael Slezak

The “extent of neglect” in the documentation of family histories, revealed by an Australian **audit**, has sparked calls for renewed emphasis on the recording of family histories.

“The time has come for family history taking to be returned to its rightful place in routine clinical examination,” the study authors wrote in the *MJA*.

Only 16% of patients admitted to a short-stay medical unit at the Royal Perth Hospital had a family history recorded with some specific details about the presence or absence of a disease in at least one relative, the retrospective audit of 300 randomly selected patient records found.

A further 10.3% had some information about family history in their records, but it was insufficiently detailed to allow others to



Renewed emphasis needed on family histories

assess it, the researchers found.

Only 5 of the 300 (1.7%) records were found to include details of two generations of family members.

Among patients presenting with chest pain, the results were more

favourable with 47% having some comment about family history recorded and 27% had more specific details about some family member.

The researchers also found a trend towards increasing family history among younger patients.

The researchers considered a number of possible causes of the “extent of neglect in documentation” found in their study, including high workloads, a focus on immediate acute issues and “local issues”.

In an accompanying editorial, three experts called for greater emphasis on family history, saying it “should not be seen as a relic of medical school teaching”.

“It is time to reconsider the clinical benefits arising from family history and start making better use of it in clinical practice,” they said.

MJA 2010; 192:682-84.

What do you think?

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In brief

Pfizer suspends tanezumab trials

Pfizer has suspended clinical trials of its experimental biotech arthritis drug tanezumab at the request of the US Food and Drug Administration after some patients on the medication experienced a worsening of their condition requiring joint replacement surgery.

According to a media statement, the halt includes suspension of recruitment of new patients and the dosing of existing patients in the Pfizer osteoarthritis program.

The suspension follows the presentation, by Pfizer, of positive data on tanezumab at the EULAR conference in Rome last week, which showed that it significantly reduced knee pain versus placebo in patients with osteoarthritis, the statement said.

The US FDA has asked Pfizer to present its assessment of the potential implications of the adverse events for its other tanezumab clinical programs.

No need for orthopod follow up



Routine post-operative follow-up of patients who have joint replacements should be abandoned because it is wasteful of time and resources and few arthroplasty failures are detected in this way, a study by orthopaedic surgeons at the Royal Brisbane and Women’s Hospital has concluded. *Australia and NZ Journal of Surgery*.

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