maximal erosion dimension in axial slices, and the results compared with 3D findings.

HR-pQCT was able to detect small (0.5 mm) erosions, with good inter-reader and intra-reader agreement and with high correlation between quantitative 3D and semiquantitative erosion scores. Furthermore, HR-pQCT images of 26 erosions from 7 patients were compared with corresponding MRI images; bone marrow oedema pattern was found to surround 20 of the 26 erosions. Similar to previous observations by Stach *et al.*,[5] and later by Fouque-Aubert *et al.*,[6] the severity of bone erosions in the HR-pQCT results was related to disease duration,[2] suggesting that bone erosions indeed reflect cumulative structural damage in patients with RA. Interestingly, higher MRI scores of bone marrow oedema correlated with structural bone damage in the HR-pQCT assessment,[2] again highlighting the importance of sites of bone marrow oedema for predicting later development of bone erosions in RA.[9] Although a tight relationship between bone marrow oedema and bone erosions has been shown in previous studies, it is interesting to find such an association when using different imaging techniques.

The study by Srikhum and colleagues[2] thus supports the value of HR-pQCT in visualizing structural damage in patients with RA. Further research from larger studies is necessary to better define the interactions between bone marrow oedema and structural bone changes. The presence of bone marrow oedema *per se* does not reflect structural damage, but rather the accumulation of inflammatory tissue in the bone marrow, which replaces the bone marrow fat. Inflammatory lesions in the bone marrow are thought to trigger cortical bone erosions in later stages of disease. Bone marrow lesions are a rich source of plasma cells; the production of autoantibodies by these cells provides a mechanistic link between bone marrow oedema and cortical bone erosions. Indeed, anti-citrullinated protein antibodies (ACPA), have been defined as major triggers of bone resorption, through induction of osteoclastogenesis.[10] The impact of ACPA in terms of structural damage has mostly been demonstrated by means of conventional radiography—it will be interesting to see whether more detailed imaging analyses such as HR-pQCT will enable better understanding of the relationship between ACPA and bone loss.

Whether and how small bone erosions affect the disease course in RA remains incompletely understood. As we have mentioned, small bone erosions can occur in healthy individuals;[4] however, as healthy young individuals do not show large lesions in HR-pQCT scans,[2] unlike patients with RA, the likelihood is that small erosions result from prior trauma or mechanical overload in healthy individuals and are not the product of an inflammatory process. In RA, such small bone erosions could indeed play an important role in the disease process by providing a link between the bone marrow cavity and the synovial space. Such interconnection would allow trafficking of immune cells along these channels, penetrating the cortical bone and allowing faster development and progression of RA.

In summary, HR-pQCT is an interesting new imaging technology for use in patients with RA, which has great future potential. Importantly, it can be used as a sensitive instrument to validate other imaging technologies for routine use. HR-pQCT can also be used to monitor structural bone changes in patients with RA according to the disease course and anti-rheumatic treatment. Further characterization of the impact of microanatomical changes on joint function will be of seminal importance in realizing the potential of sensitive imaging modalities for the assessment of RA.

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The authors declare no competing interests.

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SURGERY

Preserving shoulder movement in advanced OA—yes we CAM!

Nikhil N. Verma and Joshua D. Harris

When nonsurgical options for osteoarthritis of the glenohumeral joint are exhausted, total shoulder arthroplasty has been well studied and carries the most predictable outcome. For patients wishing to remain active, however, proven shoulder-preserving options have been less predictable. A new study now adds to preliminary evidence supporting the complete arthroscopic management procedure.

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Glenohumeral joint arthritis is a common cause of shoulder pain and loss of function. After the hip and knee, the glenohumeral joint is the third most frequently replaced joint in patients with arthritis in the USA.[1] Although less than 10% of all joint

reconstructions in the USA are in the shoulder, over 50,000 total shoulder replacements are performed there annually, a number that has been rapidly growing over the past decade.[2] No head-to-head studies have directly compared outcomes between shoulder and hip or knee arthroplasty, but success rates are reportedly >95% with all three procedures. A study published by Millett *et al.*[3] in the March 2013 issue of *Arthroscopy* has now demonstrated successful outcomes—comprising reduced pain, improved motion and function—and 85% 2-year survivorship, using the comprehensive arthroscopic management (CAM) procedure as a non-arthroplasty alternative in patients with advanced glenohumeral arthritis.

Existing evidence to support arthroscopic management of glenohumeral arthritis (Figure 1) is inconclusive and controversial, based mostly on retrospective case series and expert opinion.[1] However, well-founded concerns regarding prosthesis durability have led surgeons to turn to arthroscopic options in younger and more active patients who are unwilling to modify post-operative activities that might hasten wear, loosening, and failure of arthroplasty. In this patient cohort, a durable joint-preserving, temporizing procedure is sought.

> “...concerns regarding prosthesis durability have led surgeons to turn to arthroscopic options...”

The success of arthroscopic procedures for shoulder osteoarthritis (OA) has mostly been restricted to patients with mild arthritic changes. In comparison with straightforward arthroscopy, however, the CAM procedure consists of a more aggressive, combined approach with the potential to work in more advanced disease and to thus preserve function that would otherwise be lost to joint replacement. Millett *et al.*[3] decided, therefore, to test the CAM approach in a young and active patient population ($n = 29$, mean age 52 years) with advanced glenohumeral OA. To this end, the investigators performed arthroscopic debridement, loose body removal, inferior humeral osteoplasty, capsular release, axillary neurolysis, subacromial decompression, and biceps tenodesis in 30 shoulders and assessed outcomes using a retrospective case series format.[3]

Despite the study design and small subject cohort, the results are encouraging and promote optimism with regard to outcomes in this difficult patient population. Using both patient-reported and surgeon-implemented outcome tools (comprising assessments of pain and satisfaction, and the ASES [American Shoulder & Elbow Surgeons], SANE [Single Assessment Numeric Evaluation], QuickDASH, and SF-12 score systems), significant improvements were observed at follow-up (at a mean 2.6 years [range 2.1–4.7 years]) in the 22 shoulders that did not progress to total arthroplasty and for which outcomes were assessed. Range-of-motion in forward elevation, external rotation in adduction and external rotation at 90 degrees of abduction were each significantly improved at 2–3 months postoperatively, by 53.8°, 32.6°, and 68.7° ($P = 0.001$, $P = 0.014$ and $P < 0.001$), respectively. Survivorship (that is, nonprogression to arthroplasty) was 92% and 85% at 1 year and 2 years following surgery, respectively, with six patients undergoing total shoulder replacement.[3]

In 2011, the American Academy of Orthopaedic Surgeons (AAOS) published an evidence-based clinical practice guideline on the treatment of glenohumeral arthritis in adults,[1] the authors of which concluded that no studies of sufficient quality exist that evaluate outcomes of arthroscopic management of this condition. Level III and level IV evidence regarding outcomes and predictors of success is, however, available, from studies that now include the work by Millett *et al.*[3] In cohorts with mean ages of 38.0–49.5 years, previous studies utilized arthroscopic debridement[4–7] and capsular release,[4] whereas Millet *et al.*[3] additionally performed inferior humeral osteoplasty, axillary neurolysis, distal clavicle resection, subacromial decompression, and long head biceps tenodesis. Together, these investigations have, for example, demonstrated statistically significant improvements in patient satisfaction,[3,4] range of motion,[5,6] Simple Shoulder Test score,[5,7] ASES score,[5] Constant score,[5,7] and pain visual analogue score.[5] Recovered motion (after the procedure and at study follow-up),[7] unipolar disease,[5,6,8,9] >2 mm joint-space width (JSW),[3–5,9] absence of inferior osteophytes,[5,9] and small or central (as opposed to large, noncentral) glenoid lesions[4,7,8] at baseline were all found to be significant predictors of improved outcome or reduced rate of conversion to arthroplasty. Overall, the rate of conversion to arthroplasty is reportedly in the range 10–22% at ≤2 years follow-up.[3–7] In addition to such primary clinical outcome studies, a 2012 expert literature review and survey of shoulder surgeons (Level V evidence) drew similar conclusions about arthroscopy with regard to improvements in clinical outcome scores, predictors of success and failure, and rate of conversion to arthroplasty.[10]

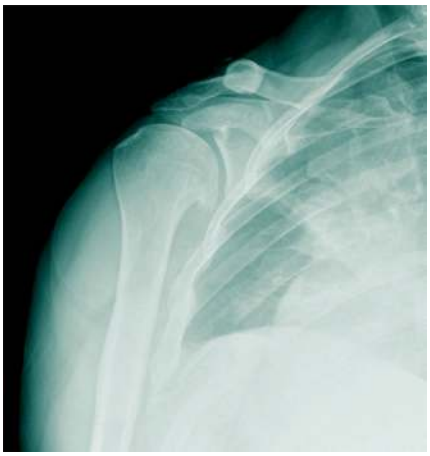

Figure 1 | Anteroposterior radiograph of the right shoulder demonstrating inferior humeral head osteophyte with minimal glenohumeral joint-space narrowing in a patient aged 50 years with moderate glenohumeral osteoarthritis. According to the study by Millett *et al.*,[3] this patient would be a good candidate for complete arthroscopic management, after the failure of nonsurgical options.

In agreement with previous findings,[4,5,9] Millett *et al.*[3] found >2 mm preoperative glenohumeral JSW to predict a better outcome of arthroscopy; indeed, patients with <2 mm JSW were 7.8 times more likely to progress to shoulder replacement and might thus benefit from undergoing arthroplasty, rather than CAM, initially. In contrast to other studies,[5,9] the investigators did not find an association between outcomes of CAM and the presence of osteophytes at baseline, or with Kellgren-Lawrence grade IV OA. The authors attribute this difference to the pain-directed procedures, including decompression of the axillary nerve, that form part of the CAM procedure as needed.[3]

With appropriate patient selection, therefore, CAM in glenohumeral OA might significantly improve patient satisfaction, pain, motion, and function. If arthroscopy is performed, the authors emphasize the importance of the combined approach, with simultaneous treatment of concomitant shoulder pathology (comprising debridement, chondroplasty of loose articular flaps, loose body removal, synovectomy, inferior humeral osteoplasty, >270° capsular release, axillary neurolysis, subacromial decompression, distal clavicle excision for acromioclavicular arthritis, and long-head biceps

tenodesis). The surgeon must be cognizant of and utilize predictors of success and failure in patient selection, caveats that apply also in the arthroscopic treatment of other arthritic joints (knee, hip, elbow, and ankle). The existence, to date, of studies mostly of retrospective design, with short-term follow-up and small patient cohorts limits the quality of the evidence supporting arthroscopic management of arthritis generally, and CAM for shoulder OA specifically, and will preclude the widespread acceptance of the recent investigation by Millett *et al.*[3] Nonetheless, the potential benefits of this procedure might outweigh the risks as a temporizing treatment in carefully selected patients.

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Competing interests
N. N. Verma declares an association with following companies and organizations: Arthroscopy, Arthroscopy Association Learning Centre, Arthrex, Athletico, Conmed Linvatec, Miomed, Mitek , SLACK Inc, Smith & Nephew and Vindico Medical Orthopaedics Hyperguide. See the article online for details of the relationships. J. D. Harris declares no competing interests.

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SPONDYLOARTHRITIS

Is it time to replace BASDAI with ASDAS?

Pedro Machado and Robert Landewé

The ankylosing spondylitis disease activity score (ASDAS) is a measure of axial spondyloarthritis (SpA) disease activity endorsed by the Assessment of SpA International Society and Outcome Measures in Rheumatology. Accumulating evidence supports the utility of ASDAS in axial SpA. So, is it time to replace the Bath ankylosing spondylitis disease activity index (BASDAI)?

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Historically, the Bath ankylosing spondylitis (AS) disease activity index (BASDAI) has been the most widely used clinical disease activity measure in axial spondyloarthritis (SpA). The BASDAI, however, is a fully patient-oriented measure that does not weight each variable, does not take into account redundancy between variables and lacks specificity for inflammatory processes.[1,2] The AS disease activity score (ASDAS)—an algorithm that combines elements of the BASDAI and patient global assessment with a laboratory measure of inflammation, either erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP) level—was created to solve some of these problems. ASDAS cutoffs for disease activity states and response criteria have been proposed and validated by the Assessment of SpA International Society (ASAS).[1] However, in the ASAS study, ASDAS cutoffs were not determined for the patient-acceptable symptom state (PASS). In a French cohort study ($n = 200$), Godfrin-Valnet *et al.*[3] investigated ASDAS and BASDAI cutoffs corresponding to the PASS, as well as those for other external standards, including flare, remission and patient-reported disease activity state (assessed as mild, moderate or severe activity).

The PASS threshold can be defined as the maximum level of symptoms with which patients consider themselves to be well. Godfrin-Valnet *et al.*[3] found that agreement between ASDAS-CRP and ASDAS-ESR was good and that values of ≤2.3 for each were associated with the PASS; these cutoffs had good specificity and moderate sensitivity. There is only one previous study investigating the ASDAS cutoff for the PASS;[4] this study suggested cutoff values between 2.5 and 3.0 for ASDAS-CRP and cutoff values between 2.8 and 3.5 for ASDAS-ESR, depending on the method used to determine the cutoff.[4] In the study by Godfrin-Valnet *et al.*[3], the BASDAI cutoff value for the PASS was 4.1; previous studies have suggested values between 3.4 and 5.3.[4]

> “...the evidence accumulated during recent years supports the replacement of the BASDAI...”

The ASDAS cutoff value for the PASS of 2.3 is slightly higher than the ASDAS cutoff of 2.1 proposed by ASAS to distinguish moderate from high disease activity (of note, the ASDAS threshold between inactive disease and moderate disease activity is 1.3, and a value >3.5 denotes very high disease activity).[1] This discrepancy is not unexpected, however, as the PASS and the absence of high disease activity represent different concepts, and it has been shown that the PASS can encompass substantial levels of pain and disease activity in AS.[4,5] Thus, PASS levels might not be an adequate target of modern therapy, which aims to achieve remission and low disease activity states. This is one of the reasons why ASAS chose more ambitious external constructs when determining their ASDAS cutoffs for the two lowest disease activity states of the scale: patient and physician global assessment <3 (measured on a 0–10 scale) were used to benchmark ‘moderate disease activity’, and ASAS-defined partial remission as well as patient and physician global assessment <1 were used to benchmark ‘inactive disease’. Furthermore, PASS cutoffs might be influenced by the way the PASS question is formulated, by the methodology used in the identification of cutoffs, by ethnicity, by socioeconomic status and by other factors