Treatment of shoulder pain

Hay and colleagues concluded, in their extended report of a trial of physiotherapy and injection for unilateral shoulder pain that physiotherapy and local steroid injection are of similar effectiveness.1 They suggest that “...high overall success rates...argue against the need for further exploratory trials in this condition”. I disagree. A large number of studies of shoulder pain have been bedevilled by diagnostic criteria that are not precise, and this study must unfortunately join the others.

Unilateral shoulder pain has a number of different causes. The study by Hay excludes a few specific conditions—in particular, a ruptured rotator cuff, but must by definition include a heterogeneous group of problems that are in fact quite discrete. These include frozen shoulder (adhesive capsulitis), rotator cuff injuries without full rupture, subacromial joint arthritis (sometimes known as subacromial bursitis), bicipital tendinitis, acromioclavicular joint disease, and subdeltoid bursitis. It is barely credible to imagine that several of these could be successfully treated by a steroid injection into the subacromial joint. In particular, the subacromial joint does not communicate with the glenohumeral joint unless the rotator cuff is ruptured, so frozen shoulder cannot be treated with a subacromial injection. Thus any study of shoulder pain must separate the different causes into different groups. Others have done this and shown that the relative benefits of physiotherapy and injection may be different.

Furthermore, it must be clear that any clinicians contributing to a trial are working to the same diagnostic criteria. Even experienced consultant rheumatologists cannot agree on exact diagnoses, as I and colleagues have shown previously, and we concluded “...in patients for studies of the treatment of shoulder lesions requires care to avoid selection of a heterogeneous group”. Given the variability of rheumatology training and experience in general practice it is unlikely that diagnostic precision will be sufficient in that setting.
Seronegative antiphospholipid syndrome


Authors’ reply

We agree with Hughes and Khamashita that the use of the term “seronegative antiphospholipid syndrome (APS)” is useful in clinical practice.1 However, the analogy with seronegative rheumatoid arthritis and antinuclear antibody (ANA) is not correct. The current criteria for the classification of rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE) allow the diagnosis of RA or SLE to be made even if the rheumatoid factor or the ANA is negative, and therefore, seronegative RA or SLE patients are included in the classification criteria and as such are not separate entities.2

Ibandronate and prevention of postmenopausal osteoporosis

Stakkestad et al reported a clinical trial where intravenous (IV) ibandronate injections, given every 3 months during 1 year, produced a dose dependent gain in mean (SD) lumbar spine bone mineral density (BMD) compared with placebo in prevention of bone loss in postmenopausal women.3 The treatment was then proposed as an alternative to oral bisphosphonates and hormonal therapy in preventing postmenopausal osteoporosis. The primary outcome was the relative change from baseline in lumbar BMD after 2 years of treatment tested by analysis of

References


Authors’ reply

We take Dr Jawad’s points and agree fully. We also believe that classification criteria are too often wrongly used in diagnosis. Our aim in writing the leader was to highlight what we believe to be a not uncommon diagnostic situation—the patient with many of the features of the syndrome in whom tests remain stubbornly negative.

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Reference

variance, with treatment group and stratum as independent variables. We found several limitations.

Firstly, the initial study was planned for 2 years, but was stopped at 12 months because of the interesting results of the IV ibandronate pivotal phase III fracture study. It was possible to infer that results obtained from a trial on fracture occurrence would apply to the study of BMD.

Secondly, given the limited changes in BMD, it could be suggested that they might be due to random variability in the procedure. The least significant change (LSC) in BMD is the percentage of change that is unlikely to be due to the precision error of the procedure. The LSC is 2.8-fold the precision error of the procedure on a specific device, site of measurement, and number of measurements. Precision error was not evaluated in the study. The sample size was calculated with an expectation of a significant change in lumbar spine BMD of 3% compared with placebo after 2 years of treatment. Would a 3% change be clinically relevant according to the variability of the measure?

Thirdly, analysis of variance was used for a dose-response model. Multiple comparisons were performed without using post hoc tests which would have adjusted the nominal threshold. The Type I error would have been lower than 0.05. Keeping these limitations in mind, on a clinical ground, IV ibandronate still needs to prove its efficacy on BMD gain.

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References

Author’s reply
On behalf of my fellow authors, I welcome the opportunity to respond to the points raised in the letter by Milka Maravic and Paul Landais. Their letter proposed several limitations of our recently published study, which investigated the efficacy and safety of intermittent intravenous (IV) ibandronate (0.5 mg, 1 mg, and 2 mg) injections in the prevention of postmenopausal osteoporosis.

In their first comment, Maravic and Landais question why the suboptimal findings from the IV ibandronate pivotal phase III fracture prevention trial led to our study being stopped prematurely after 1 year. In that trial, 0.5 mg and 1 mg IV ibandronate injections every 3 months failed to reduce vertebral fracture risk significantly, relative to placebo. The decision to stop our study prematurely was taken by the sponsors, Hoffmann-La Roche Ltd, because of ethical concerns; it was decided at the time that patients should not continue to receive treatment potentially associated with sub-optimal efficacy.

However, upon analysis of the results from the fracture prevention study, it became evident that the suboptimal antifracture efficacy was due to under dosing and not higher doses would probably provide the expected gains in bone mineral density (BMD) and decreases in biochemical markers of bone turnover. The proposition that higher doses of ibandronate would produce optimal efficacy is supported by the results from a study that evaluated the dose-response relationship associated with intermittent IV ibandronate injections in postmenopausal women with osteoporosis. In that study, a 2 mg dose of ibandronate provided significantly greater BMD increases and suppression of bone resorption markers than the 1 mg dose investigated in the fracture prevention study.

The available results from our study were analysed to enable further evaluation of the dose-response relationship of intermittent IV ibandronate. As reported, although our study was terminated prematurely, sufficient results were collected for the analysis of data for the first year of the study. These results support those seen previously: intermittent IV ibandronate produced a dose dependent effect on BMD and biochemical markers of bone resorption, with the highest dose producing the greatest efficacy. Owing to the findings from our study, together with those from prior studies, a large non-inferiority trial is continuing to determine whether a higher dose (3 mg every 3 months) or a shorter dosing interval (2 mg every 2 months) of IV ibandronate provides equivalent therapeutic efficacy relative to an oral daily ibandronate regimen, which has proved antifracture efficacy (the Dosing IntraVenous Administration study).

In their second observation on our study, the authors imply that the observed changes in BMD might have been due to the random variability associated with the procedure. However, all the BMD measurements in our study were centrally assessed by Synarc (Ballerup, Denmark), where they were also quality assured. Synarc is a well respected company that employs radiologists who are leading experts in clinical trial radiology. Thus, any variability in the assessment of spinal BMD in our study would have been kept to a minimum and the precision error (PE) associated with the measurements would be unlikely to have exceeded 1%.

Hence, as defined by several authors, including Gluer et al, the percentage of BMD change that is unlikely to be due to the variability (PE) of these measurements (the least significant change) is expected to be about 2.8% (1% multiplied by 2.8). As a result, the sample size calculations used in our study, which were based on an expectation of a significant change in lumbar spine BMD of 3% versus placebo after 2 years of treatment, were justified. Because the 3% change exceeds the 2.8% variability of the measurement procedure, it is considered clinically relevant.

In their third comment, the authors correctly state that analysis of variance was used to investigate the efficacy of the three ibandronate regimens. They go on to advise that because multiple comparisons were performed, adjustments should have been made to ensure the maintenance of the overall type 1 error. However, only three tests were performed for the primary end point (the relative effect of treatment on lumbar spine BMD), all of which were highly significant, it was not deemed necessary to apply adjustments.

Additional analyses were conducted to determine the influence of baseline spinal BMD and/or time since menopause on the primary end point. In retrospect, as a number of comparisons were performed when conducting these secondary analyses, we agree with Maravic and Landais that relevant adjustments would be optimal. Thus, we have retrospectively applied a Bonferroni correction, a well recognised test to adjust for multiple comparisons. Despite the highly conservative nature of such statistical adjustments, when the number of repeated tests is >5, only two results changed (table 1).

Therefore, reassuringly, the findings observed after applying the Bonferroni correction were similar to those reported in our publication.

In summary, although our randomised, double blind, placebo controlled study was prematurely discontinued, sufficient 1 year data from almost 600 patients were available to allow an adequate assessment of the efficacy of IV ibandronate injections in postmenopausal women without osteoporosis. We found that ibandronate dose dependently prevents postmenopausal bone loss, even when considering the variability associated with measuring BMD, and even when adjusting for multiple, repeated statistical assessments. Of note, the highest dose of ibandronate produced gains in BMD that were comparable to those observed with other therapeutic agents in this setting.

Table 1 Summary of the effect of ibandronate on mean lumbar spine BMD, relative to baseline, after 12 months of treatment (%)

<table>
<thead>
<tr>
<th>Stratum</th>
<th>Time since menopause (years)</th>
<th>Placebo</th>
<th>0.5 mg</th>
<th>1.0 mg</th>
<th>2.0 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>T score</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥1</td>
<td>1</td>
<td>1-3</td>
<td>-0.0</td>
<td>1.1</td>
<td>1.1</td>
</tr>
<tr>
<td>&lt;1, ≥ -1.99</td>
<td>-2.5</td>
<td>1-3</td>
<td>0.0</td>
<td>0.7</td>
<td>1.4†</td>
</tr>
<tr>
<td>≥1</td>
<td>3</td>
<td>3-6</td>
<td>0.4</td>
<td>0.4</td>
<td>1.9†</td>
</tr>
<tr>
<td>&lt;1, ≥ -1.99</td>
<td>-2.5</td>
<td>3-6</td>
<td>-0.2</td>
<td>0.2†</td>
<td>1.4**</td>
</tr>
</tbody>
</table>

*Difference between active group and placebo was significant (p<0.05), even when a Bonferroni correction was applied; †According to an ANOVA test, the difference between active group and placebo was significant (p<0.05). Significance was not achieved when a Bonferroni correction was applied.
We would have liked to see the antiphospholipid antibody (aPL) titre of the 11 patients and how they ranked if entered in their regression model in table 3.

A previous study identified IgG anticardiolipin antibody (aCL) as an independent predictor of carotid IMT, suggesting a possible dose effect of IgG aCL on IMT. Therefore aPL titres at the lower end of the medium range (20–80 GPL) may be less atherogenic than those at the higher end of the same range.

To confound matters further, most of their patients with primary APS had other significant conventional risk factors for atherosclerosis, whether in isolation or combination. The prevalence of hypertension, hyperlipidaemia, and obesity was 36%, 54%, and 25%, respectively, in their cohort with several patients having more than one risk factor.

We question the author’s conclusion of atherosclerosis in primary APS. As it stands, their scanning technique overestimates IMT readings, and other conventional cardiovascular risk factors are overrepresented in their cohort, potentially masking the atherogenic potential of aPL.

References

Authors’ reply
We thank Dr Ames et al for their interest in our study and for their comments about our methodology and findings of atherosclerosis in primary antiphospholipid syndrome (APS).

We agree about the difficulties of anterior carotid wall measurements. Therefore our intima-media thickness (IMT) measurements were performed on the posterior wall, as is shown in fig 2B. Our patients did not have atherosomatic plaques, consequently this artefact did not alter the IMT measurement. The colour and power Doppler ultrasound were standardised in our hospital according to the method of Cantús-Brito et al. Briefly, the sensitivity and specificity of the method were 91.3 and 92.7%, respectively.

A composite measure that combined the maximal common carotid artery IMT and maximal internal carotid artery IMT was obtained by averaging these two measurements after standardisation with subtraction of the mean and division by the standard deviation for the measurement.

Colour Doppler cannot define the “white line”, but this technique does define the intimal-luminal interface. The position of the callipers in fig 2B is correct for IMT measurement, but in fig 3B the callipers are showing the indentations in the lumen vessel.

The carotid artery IMT in our controls was 1.2 (0.44) mm. This value is higher than that reported in other studies but, however, only 7/28 controls had an IMT > 1 mm. The mean IMT in these seven controls was 1.65 mm (range 1.1–2). The explanation of these results is that the controls also had other cardiovascular risk factors and they were hospital workers. In other studies performed in Mexico, IMT was detected in more than 60% of people from the general population.

In our regression model we did not include the titres of anticardiolipin antibodies, because we did not determine them simultaneously with the carotid artery IMT study. We have only historical data of antiphospholipid antibodies. We are currently analysing the association of antiphospholipid antibodies and other new risk factors with carotid artery IMT.

In relation to conventional risk factors for atherosclerosis, we agree that these are confusing variables, but the logistic regression analysis controlled for these factors. Certainly, it is very difficult to have patients and controls who do not have traditional risk factors for atherosclerosis.

In view of all the above mentioned information, we think that our patients with primary APS had a significantly greater IMT of the carotid arteries than the controls.

The results are not overestimated because all ultrasound scanning was performed under similar conditions in both groups, by a single experienced radiologist who was unaware of the clinical information, and using the same sonograph equipment.

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The results are not overestimated because all ultrasound scanning was performed under similar conditions in both groups, by a single experienced radiologist who was unaware of the clinical information, and using the same sonograph equipment.

References
Inflammatory rheumatic disorders

I was interested to read the paper by Guillenim et al where the authors used a detailed questionnaire for a telephone survey of patients to assess rheumatic problems. They have used a similar but somewhat more extensive questionnaire, which was initially designed for doctors such as general practitioners to complete and return through my cyberclinic project.

In this study we evaluated the feasibility of a rheumatology consultation and advisory service using internet and email. We devised and posted a detailed rheumatology pro forma on our website (www.cyberrheum.org) accessed 26 February 2004 for general practitioners to complete and return on the internet. Two trainee doctors interviewed and completed the pro forma for 207 new patients. Based on this information from the questionnaire alone we were then able to provide provisional diagnoses and management plans, which we compared with those drawn up after a face to face assessment of the same patients in the outpatient clinic. Diagnostic concurrence was noted in the majority of patients (86%), no changes in further investigations, such as radiographs and blood tests, in a sizeable proportion of patients (62%) were necessary, and the majority of patients (74%) required no changes in the treatment plans suggested.

Our results show that it is quite feasible to offer an internet based outpatient consultation and advisory service in rheumatology and possibly other disciplines. Perhaps it is more practical in rheumatology because most of the diagnostic clues lie in a detailed history taken from the patients (as in the patient questionnaire we devised), only then aided by clinical examination and laboratory tests.

To our knowledge this was the first and only such internet-based service available to general practitioners. This, when established, can enable centres to streamline referral processes and provide early diagnosis and management plans for patients, even before their first appointment, which then works as a review appointment (as all the results and details of the patients are available when first attending the clinic). As a result we can cut the consultation time in the clinic to half the usual time required, a consequence of which is that we can see more new patients and perhaps also see more review patients.

We noted that general practitioners were finding it difficult to complete the pro forma on the website with the patients. Therefore, subsequently we devised a patient completed questionnaire, available on the same website, so that patients could complete this pro forma at home and bring it to the GP surgery with the help of an assistant such as a nurse. This facilitates referral and reduces demand on general practitioners’ time. We have been able to devise a separate questionnaire, again posted on our website, to facilitate referrals relating to osteoporosis services, and reduce our response time to management plans.

Although this is certainly of interest for the consultation and care of patients, a prevalence survey using telephone based technology. This is not only because people have more access to telephone than internet and email but also, above all, because people in the general population, the target of such a survey, including people with and without disease, will not spontaneously answer an internet based questionnaire. Perhaps it is more practical in rheumatology because most of the diagnostic clues lie in a detailed history taken from the patients (as in the patient questionnaire we devised), only then aided by clinical examination and laboratory tests.

We are therefore not surprised that the authors found a similar but somewhat more extensive questionnaire, which was initially designed for doctors such as general practitioners to complete and return through my cyberclinic project.

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We appreciate Dr Pal’s presentation of a rheumatology consultation and advisory service posted on a website for use by general practitioners, further completed with patient’s targeted online questionnaire to facilitate telephone based service available to general practitioners. When established, this could enable centres to streamline referral processes and provide early diagnosis and management plans for patients, even before their first appointment, which then works as a review appointment (as all the results and details of the patients are available when first attending the clinic). As a result we can cut the consultation time in the clinic to half the usual time required, a consequence of which is that we can see more new patients and perhaps also see more review patients.

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References


Mycobacterial knee infection in patients with idiopathic inflammatory myositis

We read with interest the report by Callaghan and Allen about a patient with inflammatory myositis who developed Mycobacterium malmoense infection in her right knee. The authors state in their discussion that they reviewed the literature and found no cases of isolated joint infection with this organism. However, in this journal, we have previously reported three patients with inflammatory myositis who developed mycobacterial knee infections, one of which was with M malmoense.

It is interesting to see that another case of this infection with this rare organism has been described in a patient with inflammatory myositis. We suggest that it is sensible to ensure that synovial fluid aspirated from joints of patients with autoimmune rheumatic disease receiving long term immunosuppressive drugs is sent specifically for mycobacterial culture. Possibly, some atypical mycobacterial infections are being undiagnosed. Such infections could become more common with the increasing use of biological therapies for autoimmune rheumatic disease.

References


Author’s reply

Apologies for not picking up this interesting article in our literature search. Drs Isenberg and Haq are, of course, right. Thank you for letting us know.

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morphea with longstanding ulcerations of skin or scars are at risk for the development of squamous cell carcinoma. In a recent overview we found seven case reports on squamous cell carcinoma in patients with panniculitis or generalised morphea. The number seems remarkably high because this disease is extremely rare. The observation has not only some impact on follow up but also on treatment as well. The use of ultraviolet based treatments like UVA1 or PUVA (psoralen plus UVA) widely used successfully for more severe or widespread forms of morphea might further increase the risk of squamous cell carcinoma in these patients.

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Correspondence to: Dr U Wollina; wollina-uw@khdf.de

References

Author’s reply
Dr Wollina’s observations are of great clinical and research interest. Unfortunately, we excluded patients with localised morphea in our study owing to incomplete ascertainment in South Australia. We agree that further investigation of this interesting area is needed, particularly in relation to the influence of treatment. However, epidemiological studies of rare diseases are a challenging area of research.

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Correspondence to: Dr C L Hill; catherine.hill@nwahs.sa.gov.au

doi: 10.1136/ard.2003.018429

Reference

FORTHCOMING EVENTS

International Congress on SLE and Related Conditions
9–13 May 2004; New York, New York, USA
Contact: Dr C L Hill; hill@medsci.ox.ac.uk
Website: www.lupus2004.org

IFOWorld Congress on Osteoporosis
14–18 May 2004; Rio de Janeiro, Brazil
IOF awards are available for scientists:
IOF C. Christiansen Research Fellowship: 45 000
IOF Servier Young Investigator Fellowship: 40 000
Contact: Congress Secretariat at info@osteofound.org
Website: www.osteofound.org

International Society for the Study of the Lumbar Spine
31 May–5 June 2004; Porto, Portugal
International Society for the Study of the Lumbar Spine, 2075 Bayview Avenue, Room MG 325, Toronto, Ontario, Canada M4N 3M5
Tel: 00 1 416 480 4833
Fax: 00 1 416 480 6055
Email: shirley.fitzgerald@sw.ca

8th EULAR Sonography Course
7–9 June 2004; Berlin, Germany
Organising Committee: Marina Backhaus, Wolfgang Schmidt
Contact: Congress Organisation: Gedel Congress Service
Tel: +49-30-22488390
Fax: +49-30-22488389
Email: gedel.cm@1-online.de
Website: www.eular.org

EULAR 2004
9–12 June 2004; Berlin, Germany
Contact: EULAR Secretariat
Tel: +41 1 383 96 90
Fax: +41 1 383 98 10
Email: secretariat@eular.org
Website: http://www.eular.org

First European Course: Capillaroscopy and Rheumatic Diseases
10–12 September 2004; Genova, Italy
Contact: Scientific Secretariat: Professor Maurizio Cutoio, Division of Rheumatology, DIMI, University of Genova, Italy
Email: mcutoio@unige.it
Organising Secretariat: Michela Civelli, EDRA spa, Viale Monza, 133 – 20125, Milan, Italy
Tel: +39 02 281 72300
Fax: +39 02 281 72399
Email: edracongressi@dsmedigroup.com

Xth International Conference on Behcet’s Disease
27–31 October 2004; Antalya, Turkey
Contact: Congress Secretariat, Figur Congress and Organization Services Ltd, STI, Ayazmadereci Cad. Karadut Sok. No: 7 80888 Dikilitas, Istanbul, Turkey
Tel: +90 (0212) 258 6020
Fax: +90 (0212) 258 6078
Email: behcet2004@figur.net
Website: www.behcet2004.org

4th International Congress on Autoimmunity
3–7 November 2004; Budapest, Hungary
Deadline for receipt of abstracts: 20 June 2004
Contact: 4th International Congress on Autoimmunity, Kenes International—Global Congress Organisers and Association Management Services, 17 rue du Cendrier, PO Box 1726, CH-1211 Geneva 1, Switzerland
Tel: +41 22 908 0488
Fax: +41 22 732 2850
Email: autoim04@kenes.com
Website: www.kenes.com/autoim2004

8th EULAR Postgraduate Course in Rheumatology
28 November–3 December 2004; Prague, Czech Republic
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Osteoarthritis Research Society International
2–5 December 2004; Chicago, USA
Contact: 17 000 Commerce Parkway, Suite C, Mt Laurel, NJ 08054, USA
Email: oarsi@oarsi.org
Tel: +1 800 856 439 1385
or visit http://www.oarsi.org

Vth European Lupus Meeting
3–5 March 2005; Royal College of Physicians, London, UK
Contact: Julia Kermode, Conference organiser of the British Society of Rheumatology
Email: Julia@Rheumatology.org.uk

Future EULAR congresses
8–11 June 2005; EULAR 2005; Vienna, Austria
21–24 June 2006; EULAR 2006; Amsterdam, The Netherlands

Future ACR meeting
16–21 October 2004; 68th Annual Scientific Meeting; San Antonio, Texas

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