PostScript

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Differences in the management of shoulder pain between primary and secondary care in Europe: time for a consensus

We read with great interest the articles of Van der Windt and Bouter[1] and Hay *et al*.[2] There is no doubt that the study of Hay *et al* is well designed and has practical implications. They showed that physiotherapy or subacromial joint injection are equally effective for shoulder pain. This is new evidence as, so far, there has been little evidence to support the effectiveness of any common intervention for shoulder pain.[3] However, the definition of ''shoulder pain'' illustrates the practical problem in diagnosis that general practitioners and hospital specialists face in routine clinical practice. We agree that the positive outcome for physiotherapy may reflect the increased contact time between physiotherapist and patient or the better understanding of the anatomical problem by the physiotherapist. The differences in management and in the effectiveness of physiotherapy by the British compared with the Dutch may also represent a cultural difference between the expectations and beliefs of patients in the two countries. It is likely that physiotherapy departments could be overloaded with referrals from primary care doctors if they are always the first next step in the pathway of managing shoulder problems. Hay *et al* did not carry out a cost-benefit analysis of the different treatments for shoulder pain (that is, injection *v* physiotherapy). A course of physiotherapy would cost around £200–320 (€284–454), whereas an injection would cost around £60 (€85).

There is a lack of consensus in the UK about the exact role of the general practitioner in the treatment of shoulder disease.[4] A survey among rheumatologists and physiotherapists practising in the Southeast Thames Region of London (47 rheumatologists and 9 physiotherapists) showed that the management of adhesive capsulitis in secondary care varied widely. Nearly all the rheumatologists (98%) used intra-articular steroid injection, but the time, site, and frequency of injections were variable, with 72% believing that early injections are a priority. One of five rheumatologists (22%) believed that physiotherapy and mobilisation offered no benefit. Only a small number of rheumatologists (14%) believed physiotherapy to be the only means of treatment.[5] Interestingly, 90% of physiotherapists working in secondary care wanted to see patients with a frozen shoulder as early as possible before or immediately after steroid injections. However their waiting time varied considerably (range of 3 days–3 months).

Similarly, across Europe treatment of shoulder pain varies considerably between primary and secondary care.[6 7] Therefore we propose that European consensus guidelines on the management of the painful shoulder should be developed.[8 9] This consensus may be weakened by the lack of an adequate evidence base. In addition, we would suggest a third and fourth arm to future studies—steroid injection with physiotherapy and a no intervention control group.

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Dr D G Kassimos is on study leave from the Ministry of Defence of Greece.

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Author's reply

Kassimos and Panayi deal with several important issues about the management of shoulder pain in their comments on the article by Hay *et al*[1] and our leader.[2] We agree that differences in the effect of treatment between the Netherlands and England may, at least partly, reflect differences in the organisation of care, as well as differences in expectations and beliefs between the two countries. We are also aware of the lack of consensus among general practitioners, physiotherapists, and rheumatologists about the management of shoulder pain. Between primary and secondary care, especially, the differences are large. This can partly be explained by the fact that the primary care doctor is confronted with an entirely different spectrum of disease than the specialist.[3] Many patients in primary care present with signs and symptoms that are troublesome and cause worry, but are relatively benign and have a favourable prognosis. Patients referred to secondary care have been preselected by the nature and severity of symptoms, and have another prognosis, resulting in different treatment requirements.

The lack of consensus among health professionals, indeed, emphasises the need for multidisciplinary guidelines for the management of shoulder pain. Regardless of the quality of the evidence base, multidisciplinary guidelines will facilitate communication among health professionals and may optimise diagnosis and treatment of patients with shoulder pain. We suggest that the AGREE Instrument (Appraisal of Guidelines for Research and Evaluation)[4] is used in the development of any guideline for shoulder pain. This instrument includes recommendations for the description of the scope and purpose of a guideline, stakeholder involvement, rigour of development, clarity and presentation, applicability, and editorial independence.

The development of a European guideline for shoulder pain will be quite an undertaking. The authors of the EULAR guideline for the management of knee osteoarthritis indicated that there was often discordance between research evidence and the opinion of experts.[5] In this international guideline, variation across countries in healthcare delivery systems, access to health professionals, ways of funding, and attitudes towards the disease, all contributed to this discordance. The use of a Delphi system permitted consensus agreement on difficult issues, but still the applicability in individual countries may be limited. In the case of shoulder pain, it may be wise to start out with the development of national (multidisciplinary) guidelines. As yet, only a few European countries or professional organisations have developed such guidelines.

Finally, regarding the closing point by Kassimos and Panayi, we agree that there is a need for additional research comparing physiotherapy or corticosteroid injections with a no treatment control. It might be difficult or undesirable to carry out such a

trial in patients with severe pain and limitations in daily activities, but controlled trials will certainly help to establish the effectiveness and cost effectiveness of physiotherapy and injections in patients with mild to moderate shoulder pain. Future trials may also evaluate the effectiveness of combined treatment (injections plus physiotherapy).

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Exercise in juvenile idiopathic arthritis: promise or passé

We were interested in the recently published article in the *Annals* by Takken *et al*.[1] Notwithstanding their substantial work, we have a few comments pertaining to the exercise regimens in children with juvenile idiopathic arthritis (JIA).

Firstly, we did not see any information about whether the patients had ever been following an exercise protocol before they were included in the study and also whether they were prescribed a protocol afterwards. Information about these two points is important for an interpretation of the patients' results and for providing evidence about the practical implications of the study.

Secondly, when mentioning the diminished loadbearing capacity of these subjects owing to their inflammatory disease and the immune suppressive drugs, they drew attention to a study in which weightbearing exercises were shown to improve the aerobic endurance of such patients.[2] At this point, it is noteworthy to add that the myopathic effects of corticosteroids should also be remembered when exercise is prescribed. It is known that eccentric muscle contractions in normal subjects are responsible for a much greater efflux of muscle enzymes into the circulation than is caused by concentric contractions, and are associated with ultrastructural indications of damage to the muscle.[3 4] Thus in patients with JIA—where steroid use is prevalent—concentric types of exercise should preferably be prescribed. These may include simply walking, cycling, or running. However, the list of sports which can be played is endless and there is an excess of activities these—otherwise sedentary—children can be encouraged to take part in to obtain exercise.[5] In this way not only will there be an increase in their aerobic capacities but also they will encounter fewer disabilities related to muscle anaerobiosis—much more common in children who use much more energy than adults during daily activities.

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Authors' reply

We would sincerely like to thank Özçakar and Özçakar for their response.

Firstly, the patients studied did not actively participate in endurance sports activities at the time of measurement. However, some of the patients had taken part in some sports activities in the period before the disease onset, but not in the six months before our study was performed. It is known from the literature that there is a rapid diminution in fitness once training stops.[1]

We did not prescribe exercises based on the current findings. The Caltrac is a portable electronic activity monitor that measures movements in the vertical plane. It sums and integrates the absolute value of the acceleration versus time curve and derives a numerical count that is displayed on the monitor. There are no normal values for this instrument. The described data were baseline data from a randomised controlled trial for the effectiveness of aquatic exercise therapy.[2] Secondly, we did not discuss the effects of corticosteroid treatment on aerobic fitness, because only a small minority of our patients (four) had systemic juvenile idiopathic arthritis (JIA), in which steroids are the preferred treatment. In other JIA subgroups, non-steroidal anti-inflammatory drugs and methotrexate are the common treatment in our country nowadays. A discussion on the effects of drugs and inflammation on exercise capacity can be found elsewhere.[3 4]

We could not comment on the paper cited by the authors because it had not yet been published when we wrote this letter. Furthermore, we would like to add that JIA and juvenile dermatomyositis (JDM) are distinct diseases and that the exercise capacities of these patients do differ significantly, with patients with JDM being more affected than patients with JIA.[5] Therefore, the exercise prescription for patients with JIA and JDM should be different, and adapted to the individual patients needs and capacity.

Moreover, we are not aware of studies showing an anaerobiosis in muscles of patients with JIA during activities of daily living.

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Progressive multifocal leucoencephalopathy and immunosuppression

We report an immunocompromised patient with progressive multifocal leucoencephalopathy (PML), who demonstrates the usefulness and limitation of the algorithm of Warnatz *et al*[1] for investigation of patients with pre-existing autoimmune diseases and new onset neuropsychiatric abnormalities. A prerequisite for the use of this algorithm requires a high degree of awareness for infection to prevent misclassification of the underlying problem.

This 61 year old white woman had had dermatomyositis since 1996 as manifest by Gottron's papules, heliotrope rash, proximal muscle weakness, and antinuclear antibody (ANA) titre 1/1280 speckled pattern. Previous management included azathioprine, methotrexate, hydroxychloroquine, and intravenous immunoglobulin; the disease was controlled for the previous 20 months while receiving cyclophosphamide 100 mg and prednisone 5 mg daily.

One week before admission the patient developed dizziness, weakness, and left sided hearing loss. Meclizine was prescribed for possible Ménière's disease. Facial weakness and dysarthria developed. A physical examination showed left sided hearing loss, left facial droop, left hemiparesis with concomitant graphaesthesia, and impaired stereognosis; left patella hyperreflexia was also present. Magnetic resonance imaging (MRI) of the brain was performed at an outlying facility and was felt to demonstrate a subacute infarct. There was increased signal intensity in the right posterior temporal lobe measuring 4 cm in diameter without mass effect or haemorrhage, and an additional temporoparietal lesion. Punctate areas of increased signal were seen in the mid-portion of the pons (fig 1A). She was admitted for further evaluation of stroke. Laboratory data included normal complete blood counts,

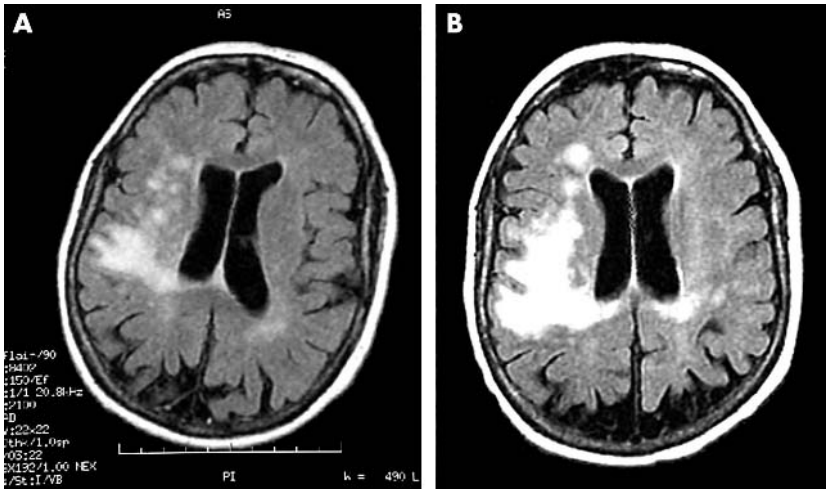

Figure 1 Magnetic resonance imaging of the brain. (A) 7 days and (B) 19 days after the initial symptoms in an immunocompromised patient with dermatomyositis and progressive multifocal leucoencephalopathy.

metabolic profile, and coagulation assays, including anticardiolipin antibodies and lupus anticoagulant. An echocardiogram and carotid Doppler ultrasound were normal.

Intensive physical and occupational therapy were prescribed. Over the next 12 days, the left sided weakness progressed. The patient also developed decreased sensation, hyperreflexia, and extensor plantar response on the left. Further evaluation was started. Cerebrospinal fluid showed 1 white blood cell/high powered field (hpf), 0 red blood cells/hpf, protein 0.43 g/l, glucose 2.9 mmol/l. A repeat MRI of the brain showed progressive changes of white matter affecting the right cerebral hemisphere, again with sparing of the cortex. Extensive involvement of the pons was present as well as minimal involvement of the right middle cerebellar peduncle. Additional cerebrospinal fluid studies included negative viral and bacterial cultures, negative paraneoplastic autoantibodies, and negative cytology. Polymerase chain reaction for JC virus was positive.

Several features of our patient's presentation are rare in PML and caused early diagnostic confusion with delay in the diagnosis. These included the acute nature of the neurological event as well as cranial nerve involvement. Ménière's disease was initially suspected owing to the sudden onset of dizziness and left sided hearing loss, and probably reflects CN VIII involvement, as MRI did not have findings to suggest a central lesion at the cerebellopontine angle. Stroke, being considerably more common than PML in immunocompromised patients, was a further consideration in this patient owing to the acute onset of symptoms and was suggested on the initial request for imaging studies. This influenced the interpretation of the MRI changes towards infarction despite predominance of white matter involvement. The more ominous diagnosis of PML was suspected after neurological symptoms worsened (12 days after hospital presentation and 19 days after the initial event). Interpretation of the second MRI was that stroke was unlikely owing to the rapid progression, distribution, and cortical sparing, and PML was likely in this immunocompromised patient (fig 1B).

PML is well reported in HIV/AIDS publications, but there are fewer than 30 cases described in rheumatology patients, resulting in a low degree of awareness. This case emphasises the importance of informing radiologists about the immune status of patients being studied so that appropriate consideration for infection may be entertained. Otherwise, this algorithm may not be used, resulting in missed or delayed diagnosis.

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Authors' reply

Dr Cuevas and colleagues express the concern that a high degree of awareness for infection is needed to prevent misclassification of early progressive multifocal leucoencephalopathy (PML). As we point out in our article, the sole risk factor for cerebral opportunistic infections is immunosuppression. The clinical distinction between PML and central nervous system involvement of systemic rheumatic diseases is always vague. Thus, in all immunosuppressed patients with a new onset or change of cerebral symptoms a careful diagnostic approach is recommended.

There is general agreement that close communication between rheumatologists and radiologists clearly helps to interpret brain images correctly.

We agree that subacute cerebrovascular disease may also be a differential diagnosis in early PML as may other diseases such as ADEM, multiple sclerosis, sarcoidosis, or multifocal glioma. The topographic pattern in PML (sparing of cortex) largely excludes large-vessel stroke, but it may be confused with subacute lacunar infarcts. Further, the neurological deficits, including cranial nerve involvement together with middle sized lesions at three typical locations, do not support the assumption of stroke. Acute onset of symptoms may occur in PML.[1] The early PML lesions are typically asymmetric and multifocally distributed in the white matter. On the other hand, acute and subacute ischaemic lesions can easily be differentiated from PML and similar lesions by diffusion weighted sequences. In later stages PML lesions are confluent and expand concentrically, strongly suggesting the diagnosis.

Cerebral vasculitis, which has been seen rarely in patients with dermatomyositis,[2 3] could be differentiated from PML by the enhancement of the lesions after administration of gadolinium, and may be excluded by the lack of disease activity.

The differential diagnosis in immunosuppressed patients with systemic rheumatic diseases and cerebral symptoms is wide. The diagnosis may be time consuming and costly. Algorithms may be helpful in this setting.

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BOOK REVIEW

The antiphospholipid syndrome II

Eds R A Asherson, R Cervera, J C Piette, Y Shoenfeld (Pp 480, $149.) Amsterdam: Elsevier, 2002. ISBN 0-4445-09879.

The Antiphospholipid Syndrome II, subtitled *Autoimmune Thrombosis,* aims to give an overview, in four parts, of this intriguing syndrome. First is a brief overview of the history and epidemiology, a second part deals with immunology and pathophysiology, a third deals with clinical features, and, finally, several chapters discuss management and prognosis of the syndrome. Each part consists of a series of topics written by authorities in the field. The separate chapters can be considered as in-depth reviews of the item discussed.

As suggested by the title, all aspects of the syndrome are highlighted. Most chapters have a structured format, are illustrated, and well referenced. References are updated to 2001. The subject index is useful and directs the reader adequately to the items searched for. The book is especially suited for such an approach because the introduction to each chapter supplies the reader with similar, general information about the APS. Moreover, various chapters overlap. The reason probably is that the chapters are somewhat heterogeneous in selecting studies and topics to be discussed, and are not always restricted to didactic overviews. For use in clinical practice the book would have gained by including diagnostic flow diagrams and discussion on differential diagnostic dilemmas. The ultimate answers of how to deal with certain clinical situations are lacking, simply because these answers are not available yet. APS is studied extensively and further insights are developing continuously, making parts of a book like this quickly outdated.

Nevertheless, *The Antiphospholipid Syndrome II* is a very valuable source for those who want to have an overview of the great progress which has been made in fundamental research, the increasing pathophysiological insights and the current treatment modalities in APS. It is particularly useful for researchers and of value for clinicians dealing with patients with APS and the various disease manifestations these patients can develop.

M Bijl, C G M Kallenberg

CORRECTION

Updated consensus statement on biological agents for the treatment of rheumatoid arthritis and other immune mediated inflammatory diseases (May 2003) (Furst D E, *et al*. Ann Rheum Dis 2003;62(suppl II):ii2–9.)

One of the authors names was incorrectly spelt. It should have been **Kavanaugh A F**.

Corrections printed in the journal also appear on the Annals website www.annrheumdis.com and are linked to the original publication.

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