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Pain in Patients not Directly Affected by the Surgical Procedure

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Abstract

Assessment of the osteoarthritis patient is primarily clinical. Although it has been suggested that the nature of the pain may be of help, i.e. nocturnal pain representing intra-osseous hypertension, in practice this is rarely of assistance.

Keywords: Clinicians; Pain assessment; Clinical trials; Pain measure; Techniques; Pain behaviours

Introduction

Duration is an important consideration since recent deterioration merits consideration of super- imposed pathology, e.g. sepsis or avascular necrosis. A marked inflammatory component is suggested by symptoms of prolonged early morning and inactivity stiffness, and signs of warmth, effusion and 'stress' pain. The examination is also directed to determining the precise site of maximal tenderness, eg joint line or periarticular. Multiple periarticular and muscular tender sites should prompt consideration of the fibromyalgia syndrome particularly if associated with sleep disturbance [1]. Finally consideration should be made of the patients affect since this may be as important as structural damage in influencing the degree of disability and pain experienced by the patient. Symptoms arising from adverse biomechanical factors are best treated biomechanically, utilising the expertise of the dietician, physiotherapist, occupational therapist, orthotist and surgeon. It is important to identify such factors since appropriate therapy is often effective and probably less hazardous than inappropriate medication. Ultimately only long-term studies in humans can demonstrate whether chondro-protective agents are beneficial. The many difficulties encountered with such trials relate to problems of long-term compliance, assessment and definition. Human osteoarthritis presents when symptomatic and is diagnosed as osteoarthritis only when structural change is evident [2]. In animal models the time of onset of the osteoarthritis process is pre-determined and early or prophylactic treatment can be employed. Meniscectomy results in premature osteoarthritis in predisposed individuals and this may be one suitable human model in which to assess chondro-protective agents. Alternatively patients with osteoarthritis at one site may, in the relative short-term, develop OA in other sites and this rate of acquisition of new sites might be a useful marker of chondro-protection.

Discussion

In spite of all the difficulties a number of attempts to study chondro-protective agents have been made. The current therapy of osteoarthritis depends on a careful approach to eliciting the source of symptoms. This relies on a careful clinical examination to localise tenderness to joint-line or periarticular sites, and to determine if a more widespread soft-tissue pain problem is present. Biomechanical factors, producing periarticular pain or mal-alignment may respond well to biomechanical treatments. In particular muscle strengthening exercises and weight reduction are often beneficial [3]. Local periarticular tender sites arising from bursitis or enthesopathy may respond to local corticosteroid injection. More widespread pain resulting in the fibromyalgia syndrome should be approached with regard to improving aerobic fitness and consideration of a trial of antidepressants. Pain, the predominant symptom should initially be treated by simple analgesics and regular and full dose paracetamol may be sufficient [4]. If there is marked synovitis, local therapy in the form of corticosteroid injections can also be effective and should be considered, though benefit may only be temporary. NSAIDs should only be used after careful assessment of risk, and consideration of the alternatives. Regular critical reassessments of continued use should be made and repeat prescribing avoided. There is currently little rational basis for preferring particular NSAIDs, but topical administration is attractive. Antidepressants may have a place in the treatment of chronic pain and chondro-protective agents may be important drugs for the future [5]. The optimal management of pain in osteoarthritis requires identification of its source. For example, much pain in osteoarthritis appears to be periarticular and appropriate local measures, e.g. periarticular steroid injections, may be effective. If analgesia is required, paracetamol should be used at maximum dosage before progressing to non-steroidal anti-inflammatory drugs. Nefopam, codeine phosphate, di-hydro-codeine or combination preparations are often used though any increase in efficacy may be offset by greater toxicity. Indeed evidence for improved efficacy in osteoarthritis over paracetamol is scanty [6]. Stronger opioids should not be used; uncontrolled pain requires re-evaluation or an alternative approach such as a regional nerve block or surgery. Local heat, such as provided by rubifacients, may be beneficial. Similarly acupuncture and transcutaneous nerve stimulation may be beneficial, though convincing evidence for either is sparse. All may act by modulating pain gating. Depression is often a feature of chronic pain and in addition many anti-depressants have analgesic properties. Surprisingly little is known of the incidence neither of depression and anxiety in osteoarthritis nor of the efficacy of anti-depressants. Osteoarthritis is the commonest affliction of synovial joints with structural joint changes of osteoarthritis, present in approximately half of the adult population. However symptoms are present in a smaller proportion depending on the joint involved [7]. Structural osteoarthritis may thus be regarded as either symptomatic or asymptomatic and optimal treatment requires

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identification as to which of many possible mechanisms are responsible for producing symptoms. The realisation that osteoarthritis is not simply a passive wear and tear phenomenon but an active process, that has been phylo-genetically preserved, and which can be associated with good functional outcome, has produced the concept of osteoarthritis as a process rather than a disease, representing the normal reparative response to joint insult. Consequently there exists the possibility of manipulating this process towards a favourable outcome. This underlies recent interest in chondro-protective agents which may maintain cartilage and thus possibly, joint function. Assessment of response to treatment involves three components. Firstly the patient's desire is for symptom relief and thus his/her self-assessment of response is paramount [8]. Secondly there is a need to maintain or improve function: a variety of instrument, are now available to assess this aspect. Thirdly, if the osteoarthritis process can be modified, then a sensitive, objective measure of response is required. Currently this involves long-term estimation of gross structural change, but ultimately more sensitive measures of structural change or biochemical markers of physiological change may prove relevant. The treatment of osteoarthritis is currently purely symptomatic. To enable rational therapy, careful clinical assessment is necessary to identify the origin of symptoms. Often, effective therapy can result from a biomechanical approach such as surgery, orthotics, physiotherapy and dieting. If drugs are required, there is little evidence that the current over-reliance on non-steroidal anti-inflammatory drugs is justified. Full dose regular paracetamol should be the first line of analgesic therapy [9]. In the majority of patients, simple analgesics are probably as effective as NSAIDs. If NSAIDs are used it is necessary to review regularly their use and to be aware of potential toxicity. Many alternative strategies of pain management such as topical preparations, intra-articular steroid injections, acupuncture, radio-synovectomy, transcutaneous nerve stimulation and anti-depressants, may be effective but their precise place in the armamentarium is not yet fully established. The realisation that osteoarthritis is not a passive wear and tear phenomenon but an active process that may be potentially modified, has led to interest in chondro-protective agents, which may beneficially affect the osteoarthritic process. To date there are no convincing data available that such agents are, in fact, chondro-protective in humans. Elucidating those that are important in an individual patient should enable targeted therapy. The usual symptom for which the patient seeks relief is pain. Cartilage is avascular and a neural and it is unlikely that symptoms arise directly from its destruction. However there are associated changes in other joint tissues which may be symptomatic. Alterations in cartilage matrix may promote crystal formation and shedding, resulting in episodes of inflammation. Pain may be further modulated by the brain and spinal cord. Other symptoms, e.g. stiffness or loss of function, must also be addressed but their underlying mechanisms are poorly understood [10]. They may however include capsular fibrosis, tendon contracture, muscle atrophy, muscle inhibition and reduced fitness. For the minority in whom NSAIDs cannot be substituted by simple analgesics it would seem wise, particularly in elderly females who may be at increased risk, to monitor for adverse effects. Although such monitoring has been widely recommended both by manufacturers and others, the optimum method has not been specified. Ideally, since acute deterioration in renal and hepatic function may occur in patients recently commenced on NSAIDs, renal and liver function should be measured prior to, and 2-4 weeks after, starting the drug. Iron deficiency anaemia may result without evidence of overt gastrointestinal bleeding and six monthly full blood counts might be appropriate. Differences in the safety profiles of different NSAIDs have yet to be established. There are many methodological difficulties including the difficulty of establishing equipotent doses, self-selection of patients in non-randomised surveys, and bias in retrospective surveys. Since NSAIDs are given for symptom relief only, the smallest dose that provides this should be used. Outcomes for patients with rheumatoid arthritis have improved over the past decade, owing to recognition of importance of a structured treat-to-target treatment protocol and of early treatment, in addition to increasing access to potent therapies. American College of Rheumatology response criteria, quality of life measures, and differences in radiographic progression have all been used as treatment endpoints to demonstrate improvement.

Conclusion

Studies clearly demonstrate that disease management protocols emphasizing tight control result in lower disease activity, more patients in remission, and less joint damage. Initiation of therapy and achieving low disease activity early in the first year of disease has a major effect on clinical and radiographic outcomes years later. Finally, initiating aggressive treatment after onset of rheumatoid arthritis and maintaining tight control is a feasible real world strategy.

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Conflict of Interest

None.

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