OsteoArthritis - The Medical Model

Osteoarthritis (OA) is the commonest of joint diseases. It involves deterioration of the articular cartilage and bone. The degree to which this can occur is very much an individual process, with age being a major factor. The principal symptom is pain and this can cause a great deal of suffering. Several theories prevail as to why the cartilage deteriorates. What appears to be the most popular theory has been discussed below. Management of OA is based largely on symptomatic treatment of pain and new innovations in this area are urgently needed.

Introduction

According to the Greeks the term 'arthritis' simply means 'inflammation of the joints' (Brewerton, 1995, p43). However, Osteoarthritis (OA) is not primarily an inflammatory disease and the term 'arthritis' can be somewhat misleading. It was not until the early part of the twentieth century that osteoarthritis was distinguished from rheumatoid arthritis, largely by microscopical analysis (Cecil and Archer, 1926, p741-746). Osteoarthritis was found, primarily, to be a result of cartilage and bone degradation. Inflammation came as a secondary matter and could take a considerable amount of time to develop (Brewerton, 1995, p125). For this reason, a lesser-known term for this condition is 'osteoarthrosis' (Kersley and Glyn, 1991, p7). None-the-less, OA can be considered a chronic inflammatory disease, especially as the more aggressive forms can progress rapidly and be accompanied by severe inflammation (Stevens and Lowe, 1995, p491).

There are several theories on the pathology of OA and how the cartilage deteriorates and then remodels itself (Hough, 1997, p1945). To attempt to address all of them is beyond the scope of this essay. Therefore, I have attempted to summarise what appears to be the one of the most popular theories at present. However, it is worth bearing in mind that the ability of the cartilage and bone to remodel and repair itself is very much dependant on the individual and that OA is a common reaction to any number of mechanical factors and injuries (McGee, Isaacson and Wright, 1992,p2084).

Epidemiology

OA occurs in about 20% of the entire population (Kumar & Clark 1994, p384), but increases significantly with age. It is estimated that the disease's incidence may be as high as 75% for those aged over 50 (Woolf 1998, p1072). Hip OA is less common in Chinese and African populations.

OA has been with us and all other vertebrates - including whales and dinosaurs – for hundreds of thousands of years. Evidence of this has been found in bone records of the past. (Beers and Berkow 1999; Inoue et al 2001, p70-73; Mills & Bone 2000, p248; Waldron, 1995 p385-389).

Studies of Asian and Caucasian human skeletons as old as 7000 years have taken place and it has been discovered that hunter – gatherers held a higher incidence of elbow and knee OA as compared to agricultural populations (Inoue et al 2001, p70-73). Physical activity in these groups undoubtedly played a part. In the present day, tibiofemoral (knee) OA has become much more common in Caucasians as compared to ancient skeletons found in Britain (Waldron, 1995 p385-389). Further investigation may determine why although it is likely that knee bending plays a large part. It is felt that more research in this area will probably help towards a better understanding of the aetiology of OA (Inoue et al, 2001, p70-73).

Aetiology

Little is really known about the aetiology of OA (Woolf 1998, p1072; Mills & Bone 2000, p248). Traditionally, it was believed to be caused by toxins accumulating at the joints, due to the hold-up in the circulatory flow around these areas (Mills and Bone, 2000, p248)

Classification

If the disease is not associated with a known cause, it is classified as primary osteoarthritis. If the OA is caused by other known factors then it is classed as secondary OA. These factors can include; pre existing joint damage, such as rheumatoid arthritis; metabolic diseases, such as chondrocalcinosis ; systemic diseases, such as haemophilia and sickle cell; and mechanical factors, such as hypermobility of the joint (Kumar and Clark, 1999, p467).

Risk factors

There are certain risk factors that need to be taken into consideration with OA. These are; ageing, load bearing, obesity, genetics and gender. Out of these, age and load bearing patterns are thought to be the most important (Souhami & Moxham 1999, p941). OA can arise from wear and tear on the cartilage and bone due to excessive and repeated load bearing on normal articular cartilage or normal load bearing on weakened cartilage. The bone attempts to repair and remodel itself, but it seems that the joints capacity to regenerate itself is more limited than that of other tissues. Secondary to this is inflammation (Woolf, 1998, p1072).

Obesity has been associated with OA of the knee (Kumar & Clark 1994, p384; Woolf 1998, p1072) and this is probably due to lack of exercise as well as increased load. Coggan et al (2001, p622-627) propose that if all overweight and obese people reduced their weight by 5 kg or until their BMI was within the recommended normal range, 24% of surgical cases of knee OA might be avoided.

In some families where OA is undeniably prevalent, studies have been carried out on their genes, and abnormalities have been found in the coding for collagen. (Woolf 1998, p1072) Another study (Keen, Hart and Spector, 1997, p1444), has found that a genetic variant of the vitamin D receptor holds an increased risk of OA of the knee.

A particular type of OA – polyarticular – is much more common in female than in males, leading to the supposition that hormones may help determine this disease (Woolf 1998,p1072). Wluka et al (2001, p332-336) have found that a decrease in cartilage volume is better correlated to the number of years after menopause has started, rather than age itself. They also propose that use of oestrogen replacement therapy for longer than five years is linked to greater knee cartilage volume and that it may protect against OA.

Pathology

Preface

Until very recently OA has been thought of as a degenerative disease (Kumar and Clarke, 1996, p385; Stevens and Lowe, 1995, p488; Mills and Bone, 2000, p248). However it is now being described as an active or metabolically dynamic reparative process (Souhami and Moxham 1999, p941; Kumar and Clarke 1999, p466). Arguably, it is still a degenerative disease but this seems like a much more positive description of the bones attempts to remodel itself.

Articular cartilage is predominantly made up of type II collagen. This provides the structural mesh. Supported in this mesh are proteoglycans and chondrocytes. Proteoglycans are polysaccharide molecules; they are vital to the function of articular cartilage, providing it with the ability to absorb load. Chondrocytes synthesise and maintain the cartilage matrix (Woolf, 1998, p1071-1073).

When healthy cartilage becomes compressed due to load bearing, fluid is pumped out of the cartilage into the joint space. It then flows into the capillaries and venules. As the joint decompresses the fluid returns back to the cartilage, allowing it to re-expand, re-hydrate and absorb the nutrients it

needs (Beers and Berkow 1999, p449). When visualising this process it seems very fragile and it becomes easier to understand why malfunctions occur. (Fig of healthy non specific joint)

Pathological Changes

OA is characterised by fibrillation in the cartilage, which leads to its deterioration. Bone erosion, eburnation and remodelling follows and then the synovium and joint capsule can start to thicken (Beers and Berkow, 1999, p449; Kumar and Clark, 1999, p466; Stevens and Lowe, 1995, p491; Moxham and Souhami,1998, p841) (Zaia, Liu, Boynton and Barry, 2000 p94-103).

Repeated load bearing will increase proteoglycan synthesis and make cartilage stiffer, but continuous decompression of a joint eventually decreases synthesis and causes necrosis (Arokoski et al, 2000, p186-198) Because of this, the joint space narrows until the two bone surfaces are exposed to each other, causing then to become eburnated. Constant friction causes a thickening of the subchondral bone plate and micro fractures appear. Osteochondrophytes develop and bony cysts then start to form. Inflammation encourages hyperplasia, which is the cause of the synovium and joint capsule thickening. Atrophy of the muscle can occur due to lack of use of the joint (McGee, Isaacson and Wright, 1992, p2086).

OA is extremely diverse but can be loosely put into four categories: (Beers and Berkow, 1999, p449; Kumar and Clark, 1999, p466; Stevens and Lowe, 1995, p491).

Primary generalised OA, which is more frequent in women and is usually associated with the development of osteochondrophytes at the finger joints. These bony formations are specifically referred to as ‘Heberdon’s nodes’ if they are situated at the distal interphalangeal joints or ‘Bouchard’s nodes’ if they are situated at the proximal interphalangeal joints. This is known as polyarticular OA.

Erosive inflammatory OA is where cartilage degradation runs a rapid course and is accompanied by severe inflammation. This is the more aggressive form of OA.

Hypertrophic OA has a slower development and osteochondrophyte formation.

Atrophic OA, where bone destruction occurs but there is no subchondral bone

response (i.e. no osteochondrophytes or bony cysts).

Pathogenesis

There is a general consensus (Beers and Berkow 1999, p449; Kumar and Clark, 1999, p446; Souhami and Moxham 1998, p941, Woolf 1998, p1072) that microscopical change within the cartilage leads to its deterioration and ability to absorb load. These changes start with a disturbance in the chondrocytes - the cells responsible for the maintenance of the cartilage. In these early stages of OA, chondrocytes undergo mitosis and increase synthesis of proteoglycan and type II collagen, which is the dominant form of collagen in articular cartilage (Woolf 1998, p1071).

The chondrocytes also stimulate osteoblasts and this causes the increase of subchondral bone formation. However, the new bone is stiffer and has less flexibility leading to micro fractures and the development of calluses. As more and more degradation takes place, the peripheral synovial cells undergo metaplasia leading to the formation of osteochondrophytes (Beers and Berkow 1999, p449). The appearance of these is often associated with the beginning of inflammation. (Woolf, 1998, p1073).

Bony cysts develop as a result of the joint fluid being pushed through the hyaline cartilage in towards the bone marrow (Beers and Berkow 1999, p449; Mills and Bone, 2000, p248). As joint decompression deals with the re-absorption of fluid into the articular cartilage, bony cysts are perhaps a result of malfunctioning joint decompression in particular.

As OA develops into the later stages, osteochondral bodies provoke the inflammatory response (McGee, Isaacson and Wright, 1992, p2086). The capillaries and venules in the synovium dilate; macrophages and lymphocytes stick to the vessel walls and eventually migrate through the increasingly permeable membrane into the synovium (Brewerton, 1995, p73). The stimulation of inflammatory cytokines within the synovium, most notably Interleukin 1 (IL-1) and Tumour Necrosis Factor (TNF), leads to an increased production of nitric oxide (NO). This occurs because inducible nitric oxide synthases (iNOS), inside chondrocytes, synthesise NO when they come into contact with IL-1 and TNF. Similarly, prostaglandins in the cyclooxygenase (COX) pathway can also be stimulated in this way, COX-2 being the inducible enzyme (Clancy, Amin and Abramson, 1998, p1141-1151). It should be noted that these events are really non-specific and are present in numerous chronic inflammatory conditions, not just within joints.

Growth factors present within the chondrocytes upregulate iNOS and this

leads to increased and continuous amounts of NO and being released. These high and sustained levels help to advance tissue injury by then decreasing synthesis of proteoglycans and typeII collagen, activating metalloproteinases and increasing apoptosis of chondrocytes (Clancy et al, 1998, p1141-1151). Metalloproteinases are enzymes such as stromelysin, collagenase and gelatinase that break down collagen and proteoglycans (Kumar and Clark 1999, p467). Together with the decrease in the synthesis of these molecules, these excessive amounts of NO cause a double assault on the foundations of articular cartilage.

An in vitro study carried out with cartilage explants has found that mechanical compression can increase NO production (Fermor et al, 2001, p729-737). Further studies are required in order to discover if these findings can be extrapolated to joint decompression. Hashimoto et al (1998, p1632-1638) have observed a strong correlation between chondrocyte apoptosis and cartilage breakdown. Indeed, the significance value was $P<0.01$. (perhaps put graph in here.) However, it is not yet fully understood if these mechanisms are linked, and if they are – which process triggers which, or if they trigger each other. There appears to be an agreed opinion by Hashimoto et al, Gibson et al and Roach et al (cited in Hashimoto et al 1998, p1632-1638) that because there are no phagocytic cells in cartilage, the apoptotic bodies are likely to remain in the extracellular matrix and contribute to the calcification of cartilage. This is commonly seen in OA and it is thought that the apoptotic bodies can secrete calcium.

Calcium pyrophosphate does frequently deposit in the synovial fluid, and it is thought that it can stimulate specific metalloproteinases, and thus induce cartilage degradation (McCarthy et al, 2001, p399-406. Recent research has identified the structure of a specific protein (HLA) crystal molecule present in arthritic joints, but more investigation is needed to establish the interactions between these and other cells (Brewerton, 1995, p236-249). However, Apoptotic bodies, calcium pyrophosphate and HLA proteins can, perhaps, be considered as endogenous toxins, these being the basis of traditional considerations in arthritis.

Diagnosis

Diagnosis of OA is usually based upon its signs and symptoms. In the early stages these are; pain – due to over vigorous exercise and morning stiffness – due to a long periods of inactivity. As OA progresses a creaking sound in the joint known as creptius can be heard and felt, Joint deformity can be seen by x-ray. Inflammation comes with the usual signs of redness, heat, pain and swelling and is most commonly found in the interphalangeal joints. Instability

occurs and in advanced cases there is a loss of function (Beers and Berkow, 1999, p450; Kumar and Clark, 1999, p468, Souhami and Moxham,1998, p492; Woolf, 1998, p1074)

Use of MR imaging has been discovered as tool for diagnosis of inflammation in OA (Fernandez-Madrid et al, 1995, p177 – 183). Radiography can be used to detect cartilage degradation although this is only really useful for advanced OA (Woolf, 1998, p1074; Kumar and Clark, 1999, p467)

These symptoms and signs are straightforward. However, in diagnosis, secondary arthritis should be considered first as a matter of course in case other disorders also need to be addressed.

Considerations in the management of OA.

OA can be a painful and crippling disease. It can deteriorate quality of life, making normal daily activity and social events difficult to participate in. The first consideration needs to be on the severity of the disease for each individual and how much help an herbalist can give to them.

As one of the primary symptoms is pain, management of this should be a priority. Diuretic, analgesic and anti-inflammatory herbs can all help to ease the symptoms (Mills and Bone 2000, p250). Mucilage containing herbs might also be considered if the patient is taking pharmaceutical NSAIDS, as coating the stomach may offer a small amount of protection against G.I irritation from these drugs. However, it is interesting to note that Beers and Berkow believe (1999, p451) that drug therapy is the least important aspect in the management of OA. As the herbs that are on offer for arthritis treat OA in a similar way to orthodox drugs this can perhaps be applied to herbal medicine as well.

The pursuit of non-drug measures such as hydrotherapy and physiotherapy could be made on the patient's behalf. This can be done via communication with the G.P. or by pursuing private therapists. The patient should also be encouraged to pursue these therapies for themselves. These will help with structural problems and pain.

Nutritional recommendations can be made. Diets that help to reduce weight will be the most useful for OA. Dietary change will also be of some help in reducing inflammation (Darlington and Gamlin, 1996, p 117).

The patient should be encouraged to look at their daily activities to see where

changes can be made to help their condition. For example, does their exercise regime or occupation exacerbate the condition? Does their household contain suitable furniture and any other aids to help them?

Considerations for holistic treatment can be made on an individual basis, by addressing emotional issues and psychological patterns. Hoffman believes (1990, p86) that the conflict seen within arthritic joints can be compared to psychological conflict. Kumara considers (1997, p37) that rigidity of joints is symbolised by psychological rigidity. Psychosocial factors can play an important part in the symptoms of OA and should be taken into consideration (Creamer, Cejku and Hochberg, 1999, p1785-1792).

Conclusion

Although there are a number of theories on OA, It seems reasonable to assume that its pathology is multidimensional and that all present theories could be thought of as viable and can interrelate. Conversely, the bones ability to regenerate and repair will be a very individual process.

The theory that enzymes are working to degrade the cartilage seems logical enough. This understanding appears be leading a new way in treatment of OA. At the moment, drug therapy is based around treating the symptoms of pain and inflammation. Now, recent understandings in the pathology of OA may help towards treating the actual process. This will hopefully be a step forward for those suffering from the more severe forms of OA.

Obesity is one of the main risk factors involved in OA and it is not a condition that is disappearing. This suggests that OA will only increase in the future. as It will mean an immense turnaround in human behaviour (in the developed world at least), to eliminate this risk factor.

Whilst so much of the human body has evolved to work with astonishing synchronicity, it appears that with the regeneration of joints – the process has not yet reached an ideal standard. This is not nature at its best! But as every individual has a different capability to regenerate, a classification of OA that takes this into consideration would be helpful. People suffering from erosive inflammatory OA should be considered to have a real disease and should be treated accordingly. People suffering from hypertrophic or atrophic OA are more likely to be simply suffering from growing old. They may be suffering from pain and stiffness, but it is insufficient to compromise daily activities.

The ever-increasing obsession with perfect health and expectations of health care can in some ways be obstructive to the management of OA. A new

attitude towards some types of OA might be beneficial. It is simply that we wear out, as we get older.

When looked at symbolically, the human race would do well to accept its imperfections.