Classification of degenerative arthritis

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It is suggested that the former division of degenerative arthritis into idiopathic types and those secondary to some disease process is no longer valid. Recent studies have indicated that abnormal concentrations of force on cartilage lead to the development of this disease. A classification is presented that is based on the assumption that the process is initiated by abnormal concentrations of force on normal cartilage matrix, normal concentrations of force on abnormal cartilage matrix or normal concentrations of force on normal cartilage matrix that is supported by bone of abnormal consistency.

Il est proposé que l'ancienne classification de l'arthrite dégénérative en types idiopathiques ou secondaires à un processus pathologique n'est plus valide. Des études récentes indiquent que des concentrations de forces anormales sur le cartilage entraîne l'apparition de cette maladie. On présente une classification s'appuyant sur l'hypothèse que le processus est amorcé par l'application de concentrations de force anormales sur une substance intercellulaire cartilagineuse normale, de concentrations de force normales sur une substance intercellulaire cartilagineuse anormale ou de concentrations de force normales sur une substance intercellulaire cartilagineuse normale qui est supportée par un os de consistance anormale.

The etiology of degenerative arthritis has long puzzled those interested in its management. Recent information, while not defining the exact mechanisms responsible, has shed considerable light on the nature of the condition.

Pathogenetic considerations

Degenerative arthritis is a focal, progressive disease that begins in articular cartilage and subsequently may affect all the tissues of the joint.[1] The process almost certainly involves enzymatic destruction of cartilage matrix, the enzymes emanating largely from the chondrocytes. The most important enzymes currently implicated are cathepsins B and D and collagenase.[1] While all of the enzyme systems responsible for matrix destruction have not yet been identified, the turnover rates of proteoglycan[1] and collagen[2] must be altered, by either decreased formation or increased degradation. In degenerative arthritis, degradation in excess of formation appears to be the most attractive hypothesis.[1]

It has also generally been recognized that physical forces are the initiating event in this interruption of matrix maintenance and that the chondrocytes probably respond adversely to abnormal concentrations of force applied to them within the cartilage. Abnormal concentrations of force on normal cartilage have long been recognized as an important factor in the development of degenerative arthritis.[3] However, since joints are continuously under some degree of loading, normal forces on abnormal or weakened matrix could be expected similarly to injure chondrocytes and hence interfere with matrix maintenance.

Radin, Parker and Pugh[4] have drawn our attention to the fact that the subchondral bone is an important shock-absorbing structure that contributes to the energy absorption required when a joint surface is loaded. Hence there may be two situations in which the chondrocytes are affected adversely by joint loading: first, that in which the matrix is normal but the subchondral bone is weakened and, second, that in which the subchondral support is stiffened and the normal cartilage matrix must bear an increased brunt of the load.[3]

Traditionally degenerative arthritis has been classified into two types — primary, and secondary to conditions that could be expected to produce abnormal concentrations of force on cartilage and hence its degeneration. Recently Stulberg and Harris,[5] after studying a large series of cases of primary degenerative arthritis of the hip, reported that some mechanical deviation could be found in most cases to account for the onset of the disease. It appears that the term "primary or idiopathic degenerative arthritis" can no longer be entertained by the medical community and that with further time and study we will eventually find that all instances of this disease are secondary to some insult to the cartilage.

Classification

The following classification of degenerative arthritis by cause (summarized in Table I) is based on the assumption that the insult, whether physical,

Table I—Classification of degenerative arthritis by cause, with examples

A. Abnormal concentrations of force on normal articular cartilage matrix
- I. Cartilage surface irregularities
  - Fractures
  - Torn menisci
  - Loose bodies
- II. Intra-articular misalignments
  - Epiphyseal injuries including epiphysiolysis
  - Dysplasias
  - Enchondromatoses
  - Discoid menisci
  - Legg–Calvé–Perthes disease
  - Neuromuscular imbalance
- III. Extra-articular misalignments
  - Inequality of leg length
  - Congenital and acquired varus and valgus deformities
  - Malunited fractures
- IV. Loss of ligamentous stability
  - Dislocation
  - Avulsion
- V. Loss of protective sensory feedback
  - Diabetic neuropathy
  - Tabes dorsalis
  - Congenital indifference to pain
  - ? intra-articular injections of steroids
- VI. Remote causes
  - Obesity
  - Occupational

B. Normal concentrations of force on abnormal articular cartilage matrix
- I. Pre-existing arthritis
  - Septic
  - Rheumatoid
  - Hemophilia
  - Ochronosis
- II. Metabolic abnormalities
  - Mucopolysaccharidoses
  - Gout
  - Chondrocalcinosis
- III. Genetic
  - Heberden's nodes
- IV. Iatrogenic
  - Chemical synovectomy
  - Intra-articular injections of steroids

C. Normal concentrations of force on normal cartilage matrix supported by stiffened subchondral bone
- Paget's disease
- Osteopetrosis

D. Normal concentrations of force on normal cartilage matrix supported by weakened subchondral bone
- Avascular necrosis
  - Fracture
  - Systemic steroid therapy
  - Alcoholism
  - Sickle cell anemia
  - Dysbarism
  - Gaucher's disease

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chemical, inflammatory or otherwise, results in abnormal concentrations of force on normal articular cartilage matrix, normal concentrations of force on abnormal articular cartilage matrix, or normal concentrations of force on normal articular cartilage matrix supported by abnormal bone. With this classification it matters not whether matrix destruction occurs, as Weightman, Freeman and Swanson[6] believe, following stress fractures of surface collagen in a matrix weakened by loss of proteoglycan and collagen, or, as others believe,[7] because the direct effect of physical force on the chondrocyte is to interrupt its normal metabolic processes or to release lytic enzymes into the surrounding matrix, which leads ultimately to matrix deficiency.

A. Abnormal concentrations of force on normal articular cartilage matrix

I. Cartilage surface irregularities

Fractures, torn menisci and loose bodies may produce surface irregularities leading to increased stress concentrations and eventual degeneration of cartilage. Post-traumatic arthritis may affect all joints, and the speed with which arthritis develops depends on the degree of intra-articular deformity. The forces across the joint are normal but, because of irregularities in the surface, areas of cartilage are under excessive loads.

II. Intra-articular misalignments

Several authors have suggested that some degree of incongruity is desirable for the dissipation of loads in human joints.[8-10] In the category intra-articular misalignments we have included types of exaggerated joint incongruity that may be based on developmental distortions of the opposing articular surfaces such as in the enchondromatoses or discoid menisci. Gross incongruities may occur in Legg–Calvé–Perthes disease because of failure of the acetabulum to accommodate the enlarged femoral head.

Similarly, the neuromuscular imbalance of cerebral palsy and other neuromuscular diseases may, by permitting weight-bearing in abnormal positions, produce irregular concentrations of force on certain areas of cartilage. While the above are instances of gross incongruity, on the other side of the spectrum protrusio acetabuli may be an example of too much joint congruity, such that the capacity of the cartilage to dissipate large loads to other areas is reduced, which leads to degeneration.

III. Extra-articular misalignments

Gofton and Trueman[11] demonstrated how abnormal stresses may occur in the hip and knee with gross inequality of leg length. The force concentrations that may develop in various varus and valgus deformities of joints of the leg, and the extra-articular misalignments that may occur in malunited fractures, will produce uneven loading of the joint surfaces with weight-bearing.

IV. Loss of ligamentous stability

Clinically and experimentally we know that chronic subluxations or dislocations may lead to degenerative arthritis, presumably because the loss of stabilizing ligaments may permit forces to build up in one segment of the cartilage surface.[12]

V. Loss of protective sensory feedback

Abnormal loading due to loss of sensory feedback occurs in diabetic neuropathies, tabes dorsalis and other situations that produce a Charcot joint (neuropathic arthropathy). It has been suggested that intra-articular injections of steroids might damage cartilage by creating a Charcot-like joint.[13]

VI. Remote causes of excess stress

Obesity will either initiate degenerative arthritis in some patients or aggravate it. In the grossly obese "fat men of the circus" the disease develops in the joints of their legs. Similarly, in certain occupations the joint may be required to sustain heavy loads continually; hence abnormal forces may be applied to the cartilage and lead to its breakdown. Baseball pitcher's elbow and shoulder are examples of occupational disease and, indeed, degenerative arthritis is more frequent in those doing heavy labour.

B. Normal concentrations of force on abnormal articular cartilage matrix

I. Pre-existing arthritis

Degenerative arthritis may follow septic or rheumatoid arthritis and hemophilia. In each of these conditions the matrix has had, for a certain period, a reduced content of either proteoglycan (N.S. Mitchell, N. Shepard: unpublished work) or collagen, which may permit a greater number of deformations per unit area of cartilage under loading; this number may exceed that tolerated by the chondrocytes, thereby further reducing matrix turnover and increasing the liberation of destructive enzymes.

II. Metabolic abnormalities

Mucopolysaccharidoses are often associated with the development of degenerative arthritis in later life. While the forces acting across the joints are probably normal, the mucopolysaccharide content and the architecture of the articular cartilage have been changed during growth. Gout and chondrocalcinosis, on the other hand, represent a degenerative process in articular cartilage in which uric acid and calcium pyrophosphate salts are precipitated into the matrix. Presumably this alters the mechanical properties of the cartilage and thus leads to degenerative arthritis.

III. Genetic factors

Stecher[14] reported a strong hereditary factor in arthritis of the distal interphalangeal joints, which produces Heberden's nodes. The forces across the joints are not abnormal in this condition and some inherited disorder of cartilage must be presumed.

IV. Iatrogenic

Mitchell, Laurin and Shepard[15] showed that chemical synovectomy with osmium tetroxide or nitrogen mustard produced necrosis in the surface and middle layers of cartilage. The matrix was rendered abnormal, so that normal loads led to destruction. Similarly, intra-articular injections of steroids will depress the rates of matrix synthesis; this in turn affects the mechanical response of cartilage to normal loading. In animals the result is degeneration of the cartilage.[16,17]

C. Normal concentrations of force on normal articular cartilage matrix supported by stiffened subchondral bone

Radin and colleagues[4] attributed to the subchondral bone important shock-absorbing qualities that protect the cartilage from damage due to peak loading impulsively applied. They suggested that this mechanism might fail with increasing stiffness in the bone resulting from healing microfractures or other causes. This would require that the cartilage absorb more energy, which would lead to its ultimate degradation. Paget's disease is well known to be associated with degenerative arthritis, and Jaffe[18] demonstrated a relation between osteopetrosis and degenerative disease. In both conditions there is considerable sclerosis of the subchondral bone though initially the overlying articular cartilage is normal.

D. Normal concentrations of force on normal articular cartilage matrix supported by weakened subchondral bone

Cruess and colleagues[19] showed that the degenerative arthritis produced in aseptic necrosis was due to collapse of the underlying bone. Such collapse occurred whenever the necrotic bone was remodelled by soft host granulation tissue. During such a stage, then, the shock-absorbing mechanism described by Radin and colleagues[4] is deficient and, indeed, normal forces will produce

permanent deformations of otherwise normal articular cartilage.

Comments

In our classification we attempted to relate degenerative arthritis to mechanisms for increased application of physical force to the chondrocytes. We purposely omitted an idiopathic category because of the strong impression that even those cases that cannot now be categorized radiologically must fall into one of our four groups.

Retrospective viewing of patients with established disease presents certain difficulties in defining etiology when the predisposing factors have been obscured by subsequent pathologic and radiologic development. This is especially true with certain subclinical dysplasias and other developmental diseases. Nevertheless, an attempt to consider degenerative arthritis as having various mechanical origins may in the future allow us to identify the etiologic agent in many more patients than we do at present.

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