

## An investigation of the frequency of co-existence of osteophytes and circumscribed full thickness articular surface defects in the knee joint

TO Alonge<sup>1</sup> and OOA Oni<sup>2</sup>

Department of Surgery<sup>1</sup>, University College Hospital, Ibadan, Nigeria., Department of Orthopaedics<sup>2</sup>, The Glenfield Hospital, Leicester, UK

### Summary

Osteophytes are osteochondral repair tissues formed usually at the margins of synovial joints in response to a more central full thickness articular cartilage defect. These defects can be managed using the autologous chondrocytes implantation technique or with composite osteochondral grafting. Materials for osteochondral grafting of circumscribed full thickness articular surface defects may be obtained from osteophytes. In this study, the frequency of co-existence of these two intra-articular lesions was studied prospectively in patients undergoing therapeutic arthroscopy for painful knee conditions. Thirty-three of 88 knees (37.5%) had full thickness articular surface defects and 23 of these (69.7%) had associated osteophytes formation.

**Keywords:** Osteophyte, articular surface defect, osteochondral grafting.

### Résumé

Le reparage des des osteophyte tont des reparages des tissus osteochondrial quo se forment generalement a la marge des articulations synnovicales en reponse a une defection de l'epaisseur du cartilage centrale. Ces defections peuvent etre traite en utilisant la technique d'implantation des chondrocytes antalogue on avec le greffage des composant osteochondral. Les materiaux des greffage osteochondral des surfaces artoculaires defectense pourraient etre obtement des osteophytes. Dans cette etude, la frequence de la loexistence de ces 2 lesions intra-articulaire avait ete etude de manience prospective chez les patients qui suivaient une arthroscopy therapeutique pour des genoux documex. Trente-trois des 88 genoux (37.5%) avaient une defection de la pleinitude de la surface articulaire, et 23 des ceux-ci (69,7%) avaient un plus des la pluritud de la surface articulaire, des formations d'osteophytes.

### Introduction

Circumscribed full thickness articular surface defects of the knee joint which is synonymous with full thickness chondral fracture, are important because they may eventually lead to osteoarthritis [1-4]. The natural repair tissue in these circumstances is qualitatively inferior to normal articular cartilage and may not be able to withstand physiological loads. Consequently, some form of treatment is usually recommended especially in young people. The methods currently regarded as most likely to be successful are autologous chondrocyteimplantation [5] and autogenous osteochondral grafting [6-8]. Materials for these procedures are usually obtained from the normal areas of the affected

joint and in so doing, new surface defects are created. These iatrogenic lesions may themselves become pathological in the future. Hence, osteophytes are now being considered as a potential source of graft materials [9,10] but, they can be used as grafts only if they were present in the diseased joint. The purpose of this study was to find out how frequently osteophytes co-existed with full thickness articular surface defects.

### Case series and methods

Patients undergoing therapeutic arthroscopic surgery of the knee for a variety of painful conditions during a six-month period from 1 September 1997 were prospectively studied. At operation, the presence and location of full thickness articular surface defects were recorded together with the presence or absence of osteophytes. To be accepted into the study group, a surface defect had to be:

- circumscribed (i.e., with surrounding margins of normal cartilage both visually and by probing);
- more than 5 mm in diameter for ease of visualization and measurement using the tip of the arthroscope probe;
- eburnated (i.e., be completely devoid of cartilage covering, exposing the subchondral bone) or
- if visually intact, have a gritty bony sensation on probing at one or more location(s).

In order to eliminate potential, obvious or known causes of osteophytes and/or defects, knees with bilateral disease, demonstrable ligamentous laxity [11,12] or generalized articular cartilage diseases [13] were excluded. Also excluded were patients in which arthroscopy was part of the definitive procedure such as a tibia tubercle transfer.

### Results

A total of 88 knees fulfilled the criteria for the study of which 33 (37.5%) had 62 circumscribed full thickness surface defects. There were 22 males and 11 females aged between 18 and 60 years (mean = 36 years). As shown in figure 1, there were 21 medial femoral condyle (33.9%) and 24 (38.7%) patello-femoral lesions. The lateral femoral condyle (LFC), the medial (MTP) and lateral (LTP) tibia plateau were less frequently affected.

Table 1 shows that in 23 of the affected knees (69.7%), osteophytes were observed at the joint margins. Figure 2 shows the location of the osteophytes in the

**Table 1:** Relationship between defects and osteophytes.

	Number	%
With osteophyte	23	69.7
Without osteophyte	10	30.3

Correspondence: Dr. T.O. Alonge, Department of Surgery, University College Hospital, Ibadan, Nigeria

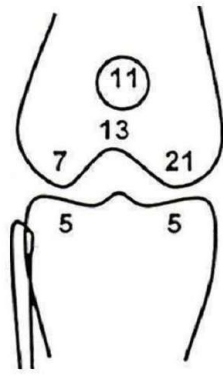


Fig. 1: Location of defects

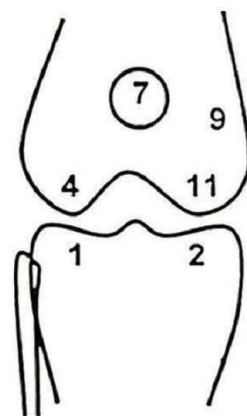


Fig. 2: Location of osteophytes

knee joints. In 5 knees (14.7%) osteophytes were located at the margins of the medial femoral condyle (MFC) alone, 3 joints (8.8%) had concomitant patella and medial trochlear marginal osteophytes and the same number had concomitant medial femoral condyle, patellar and medial trochlear marginal osteophytes. Almost 80% of the osteophytes were observed at the margins of the medial femoral condyle and the patello-femoral compartment.

#### Discussion

These results suggest that the medial femoral condyle (MFC) and the patello-femoral articulation (PFJ) are the areas of the knee most susceptible to articular cartilage defects. The reason(s) for this is not entirely clear. There are a number of factors which may be responsible for these findings. One, the excursion of the MFC is smaller than that of the LFC during flexion/extension [14] and therefore, there may be less lubrication medially. Two, there may be a relative diminution of extraosseous as well as intraosseous blood supply to the MFC compared to the LFC. Presumably this could make the MFC more vulnerable to vascular insults [15]. Three, the more rounded shape of the MFC [16] may result in a reduction in the contact area between it and the tibia plateau. As a consequence of this, there may be an increase in the contact stresses in this part of the knee compared to the lateral compartment. With regards to the

patello-femoral joint, it is practically load bearing at all times regardless of whether a person was standing, sitting or lying.

The study also reveals a close association between osteophyte formation and surface defects. There are strong experimental corroborations for this finding. Key (1931) created full thickness defects measuring 3 mm by 6 mm in the femoral condyles of mature rabbits and between 8 days and 7 months observed progressive degenerative changes in the joints with marginal osteophyte formation [1]. Fisher (1939) created full thickness cartilage defect in the central area of the articular cartilage and observed the formation of osteophytes in the lateral or marginal areas of the joint [17]. Peterson et al. (1984) created full thickness cartilage defects in the condyles of matured rabbits and observed osteophyte formation in 28.6% of defects that were not treated by autologous chondrocyte transplantation [18]. Other workers, notably Grande et al. (1989) and Outerbridge et al (1995), have also observed a strong association between full thickness cartilage defect and marginal osteophyte formation [19,20]. Thus, in clinical practice, there is a potential source in affected knees from which materials may be obtained for either chondrocyte transplantation or for osteochondral grafting [9,10]. It may not be necessary at all in most instances to obtain materials for grafting from normal areas of the knee.

The process by which surface defects stimulate osteophyte formation is not known with any certainty but the mechanisms may be speculated. The formation of a full thickness surface defect involves the necrosis of tissue and subsequent mechanical wear. It is known that the debris generated cause an inflammatory reaction. Synovitis frequently accompanies cartilage defects [21] and this is often in conjunction with synovial hyperplasia [1,17,21]. The inflammatory process may cause the synthesis of leukotrienes and other growth factors [22] which may be mitogenic for cartilage and other mesenchymal cells. Inflammation may cause an irritative metaplasia in the marginal tissues and the altered tissues may subsequently proliferate and differentiate along chondrogenic and osteogenic pathways leading to the formation of the osteochondral tissue known as osteophyte.

#### Acknowledgements

This study was supported by a grant from the British Association for Surgery of the Knee/Johnson and Johnson Fellowship. The authors wish to thank Mr. E. Olapade-Olaopa for his assistance with this research.

#### References

1. Key JA. Experimental arthritis: The changes in joints produced by creating defects in the articular cartilage. *J Bone Joint Surg.* 1931; 13: 725-739.
2. Calandruccio R A and Gilmer W S. Proliferation, regeneration and repair of articular cartilage in immature animals. *J Bone Joint Surg.* 1962; 44-A (3): 431-455.
3. Shapiro F, Koide S and Glimcher M J. Cell origin and differentiation in the repair of full-thickness defects of articular cartilage. *J Bone Joint Surg.* 1993; 75-A (4): 532-553.
4. Brittberg M, Lindahl A, Nilsson A, Ohlsson C, Isaksson O and Peterson L. Treatment of deep

- cartilage defects in the knee with autologous chondrocyte transplantation. *N Engl J Med.* 1994; 331: 889-895.
5. Pap K and Krompecher S. Arthroplasty of the knee. Experimental and clinical experiences. *J Bone Joint Surg.* 1961. 43-A (4): 523-537.
  6. Hangody L and Karpati Z. A new surgical treatment of localized cartilaginous defects of the knee. *Hungarian J Orthop Trauma.* 1994; 37: 237.
  7. Hangody L, Kish G, Karpati Z, Szerb I, Udvarhelyi I, Toth J, Dioszegi Z and Kendik Z. Autogenous osteochondral graft technique for replacing knee cartilage defects in dogs *Orthopaedics Inter. Ed.* 1997; 5 (3): 175-181.
  8. Matyas J R, Sandell L J and Adams M E. Gene expression of type II collagens in chondro-osteophytes in experimental osteoarthritis. *Osteoarthritis and Cartilage.* 1997; 5: 99-105.
  9. Oni O O A and Morrison C J. The mechanical 'quality' of osteophytes. *Injury.* 1998; 29 (1) : 31 – 33.
  10. Clancy W G, Nelson D A, Reider B, Narechania R and G. Anterior cruciate ligament reconstruction using one-third of the patellar ligament, augmented by extra-articular tendon transfers. *J Bone Joint Surg.* 1982; 64-A (3) : 352-359.
  11. Indelicato P A and Bittar E.S. A perspective of lesions associated with ACL insufficiency of the knee. *Clin Orthop Rel Res.* 1985; 198: 77-80.
  12. Harrison M H M, Schajowicz F and Trueta J. Osteoarthritis of the hip: A study of the nature and evolution of the disease. *J Bone Joint Surg.* 1953; 35-B (4) : 598-626.
  13. Oni O O A. Mechanism of injury in anterior cruciate ligament disruption. *The knee.* 1998; 5: 81-86.
  14. Reddy AS and Frederick RW. Evaluation of the intraosseous and extraosseous blood supply to the distal femoral condyles. *The Amer. J Sports Med.* 1998; 26 (3) : 415-419.
  15. Muller W. *The Knee. Form, function, and ligament reconstruction.* Berlin: Springer-Verlag. 1982.
  16. Fisher AGT. The structure and function of synovial membrane and articular cartilage. *The B M J.* 1939; 390-393.
  17. Peterson L, Menche D, Grande D, Klein M, Burmester G, Pugh J and Pitman M. Chondrocyte transplantation – An experimental model in the rabbit. 30<sup>th</sup> Annual ORS. Atlanta, Georgia. Feb. 1984.
  18. Outerbridge HK, Outerbridge AR and Outerbridge RE. The use of a lateral patellar autologous graft for the repair of a large osteochondral defect in the knee. *J Bone Joint Surg.* 1995; 77-A (1): 65-72.
  19. Bennett GA, Bauer W and Maddock S J. A study of the repair of articular cartilage and the reaction of normal joints of adult dogs to surgically created defects of articular cartilage, "joint mice" and patellar displacement. *The Amer. J Pathol.* 1932; Vol VII (4): 499 – 523.
  20. Kumar V, Cotran R S, Robbins S L. *Basic Pathology.* W.B. Saunders Company. 1992.
  21. Bennett GA, Bauer W and Maddock SJ. A study of the repair of articular cartilage and the reaction of normal joints of adult dogs to surgically created defects of articular cartilage, "joint mice" and patellar displacement. *The Amer J Pathol.* 1952; Vol. VII (4): 499-523.
  22. Kumar V, Cotram RS and Robbins SL (Ed.). In *Basic Pathology.* W.B. Saunders Company 1092.