

Microanatomic features of unilateral condylar hyperplasia

J.G.C. LUZ¹, J.R.V. DE REZENDE¹, R.G. JAEGER², V.C. DE ARAÚJO²

¹ *Department of Oral and Maxillofacial Surgery.*

² *Department of Oral Pathology.*

School of Dentistry, University of São Paulo, Brasil.

SUMMARY

Microanatomic features of unilateral condylar hyperplasia (UCH) are described. The articular surface exhibited clefts with surrounding elevations, and globules varying 0.5-2 μ m in diameter. The articular zone presented giant coiled fibers, and the proliferative zone was composed of small round cells. The findings suggest that degenerative changes occur in UCH, both in adult and juvenile forms.

KEY WORDS:

Mandibular condyle, pathology, ultrastructure.

RÉSUMÉ

Les aspects micro-anatomiques de l'hyperplasie condylienne unilatérale sont décrits. La surface articulaire montre des fissures bordées par des surélévations ainsi que des globules de 0,5 à 2 μ m de diamètre. Dans la zone articulaire on observe des fibres larges et enroulées. La zone proliférative contient des petites cellules rondes. Ces observations suggèrent que des modifications dégénératives surviennent au cours de l'hyperplasie condylienne unilatérale aussi bien dans les formes de l'adulte et que dans les formes juvéniles.

MOTS-CLÉS:

Condyle mandibulaire, pathologie, ultrastructure.

INTRODUCTION

Unilateral condylar hyperplasia (UCH) is a relatively rare condition characterized by a slow developing, progressive enlargement of the mandibular condyle, resulting in facial asymmetry and occlusal disturbances (Rushton, 1951; Sarnat & Laskin, 1979; Norman & Painter, 1980). The

etiology of UCH is obscure (Egyedi, 1969; Müller, 1979). This condition is generally seen in patients ranging from 10-30 years old, and some series have suggested that this may be predominantly a female condition (Rushton, 1951; Norman & Painter, 1980; Gray et al., 1990).

Histologically, UCH is characterized by the presence of a thickened proliferative zone, a hypertrophic fibrocartilage, and by the occurrence of cartilage rests in the cancellous bone (de Bont et al., 1985a; Gray et al., 1990). Based on histopathological criteria, classifications of active and inactive hyperplasia (Norman & Painter, 1980), and of juvenile and adult forms (Slootweg & Müller, 1986) have been proposed. No studies, however, are available in which specimens with UCH have been examined at microanatomic level.

The objective of the present study is to describe microanatomic aspects of the articular cartilage from condyles with UCH.

MATERIAL AND METHODS

Material examined consisted of two condyles, obtained during surgery from patients with UCH. The two patients were women, 36 (Case 1) and 16 (Case 2) years old, both presenting complaints of facial asymmetry and occlusal disturbances. ⁹⁹Tc bone scans of both patients revealed high activity in and around the affected joints. After dissection, condyles were rinsed in saline solution and prepared by sawing in the sagittal plane with a water-cooled steel saw. The articular cartilage remained attached to the subchondral bone. Alternate 2 mm thick samples were submitted to light and scanning electron microscopy.

Light microscopy samples were fixed in 10% formalin, and subsequently decalcified in a solution of formic acid. Seven μ m thick semi-serial sections were cut and stained with hematoxylin and eosin. Light micrographs were taken with a Jenamed photomicroscope.

Scanning electron microscopy samples were fixed in 3% phosphate-buffered glutaraldehyde and postfixed in 1% osmium tetroxide. Fragments were dehydrated in ethanol and critical-point dried using CO₂. Finally, they were sputter-coated with gold and examined with an Etec-Autoscan scanning electron microscope.

Scanning electron micrographs were obtained from alternate samples at the articular surface and the cutting surface, the different zones of the articular cartilage being evaluated in the latter.

RESULTS

Light microscopy

An identification of different zones within the articular cartilage was possible in both cases. They exhibited proliferative zone, hypertrophic chondrocytes, subchondral bone plate not closed, and cartilage rests in the cancellous bone in a large extent. Case 1 presented a discontinuous articular surface layer and a proliferative zone with a patchy distribution (Fig. 1). The fibrocartilage had a variable thickness, and clustering of chondrocytes was seen.

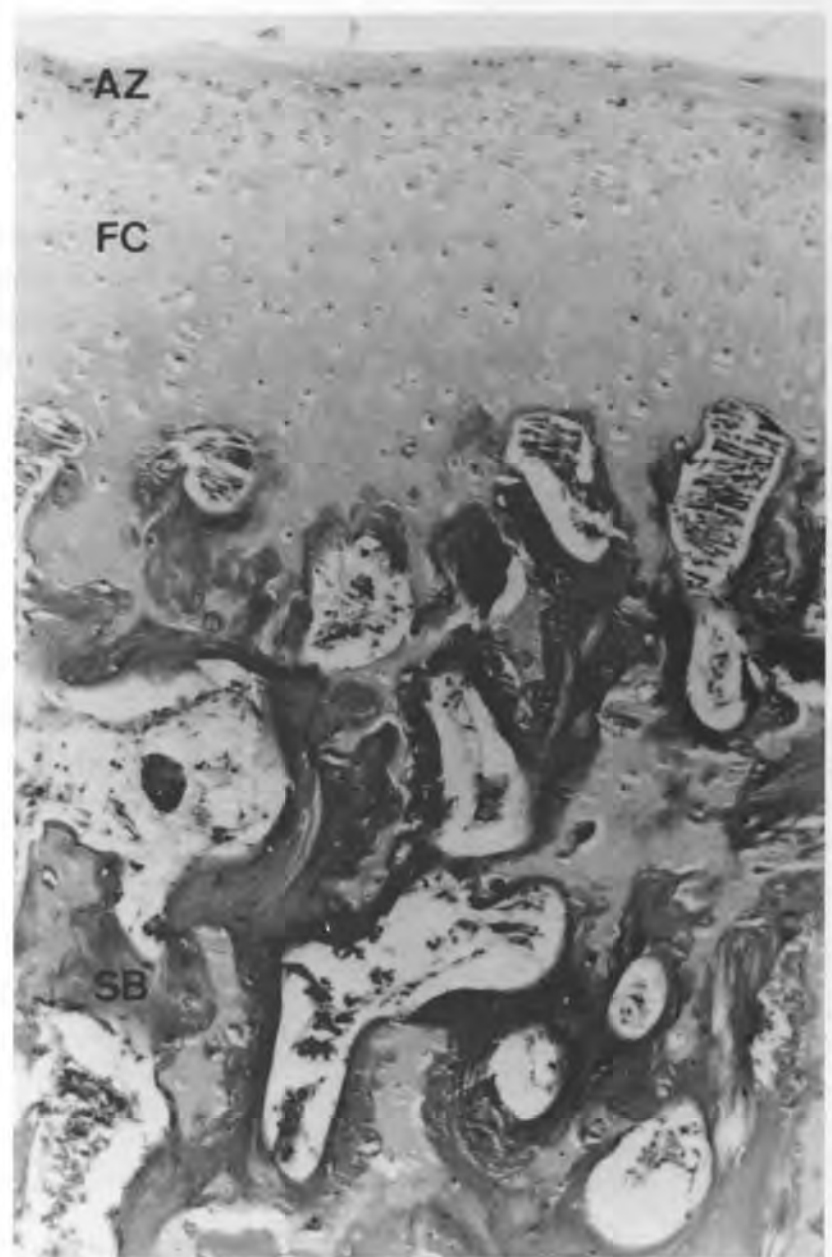


Fig. 1: Thin articular zone (AZ), irregular proliferative zone, thick fibrocartilage (FC) with hypertrophic chondrocytes, and areas of cartilage in the subchondral bone (SB) (H&E, original magnification $\times 100$).

Fig. 1: Zone articulaire fine (AZ), zone proliférative irrégulière, fibrocartilage (FC) épais avec chondrocytes hypertrophiques et territoires de cartilage situés dans l'os subchondral (SB). (H. E., grossissement: $\times 100$).

A vertical cleft, from articular zone to subchondral bone, was observed. Case 2 presented a continuous articular surface layer and a regular proliferative zone (Fig. 2). The hypertrophic cartilage layer exhibited a regular thickness. Areas of continuity of the hypertrophic cartilage and cartilage rests within the bone were frequent (Fig. 3).

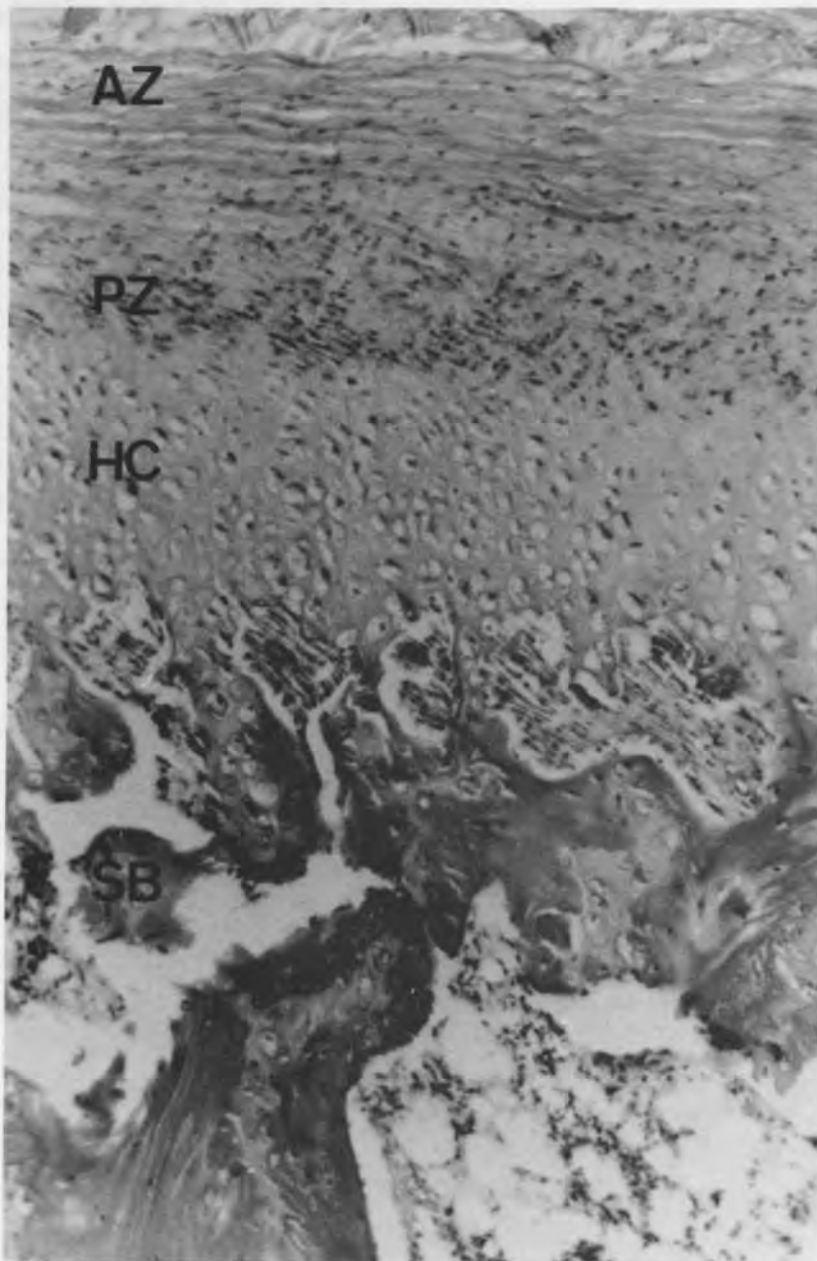


Fig. 2: Continuous and regular articular zone (AZ), proliferative zone (PZ) and hypertrophic cartilage (HC). Areas of cartilage are seen in the subchondral bone (SB) (H&E, original magnification $\times 100$).

Fig. 2: Zone articulaire (ZA) continue et régulière, zone proliférative (PZ) et cartilage hypertrophique (HC). Des territoires de cartilage sont observés dans l'os subchondral (SB). (H. E., grossissement: $\times 100$).

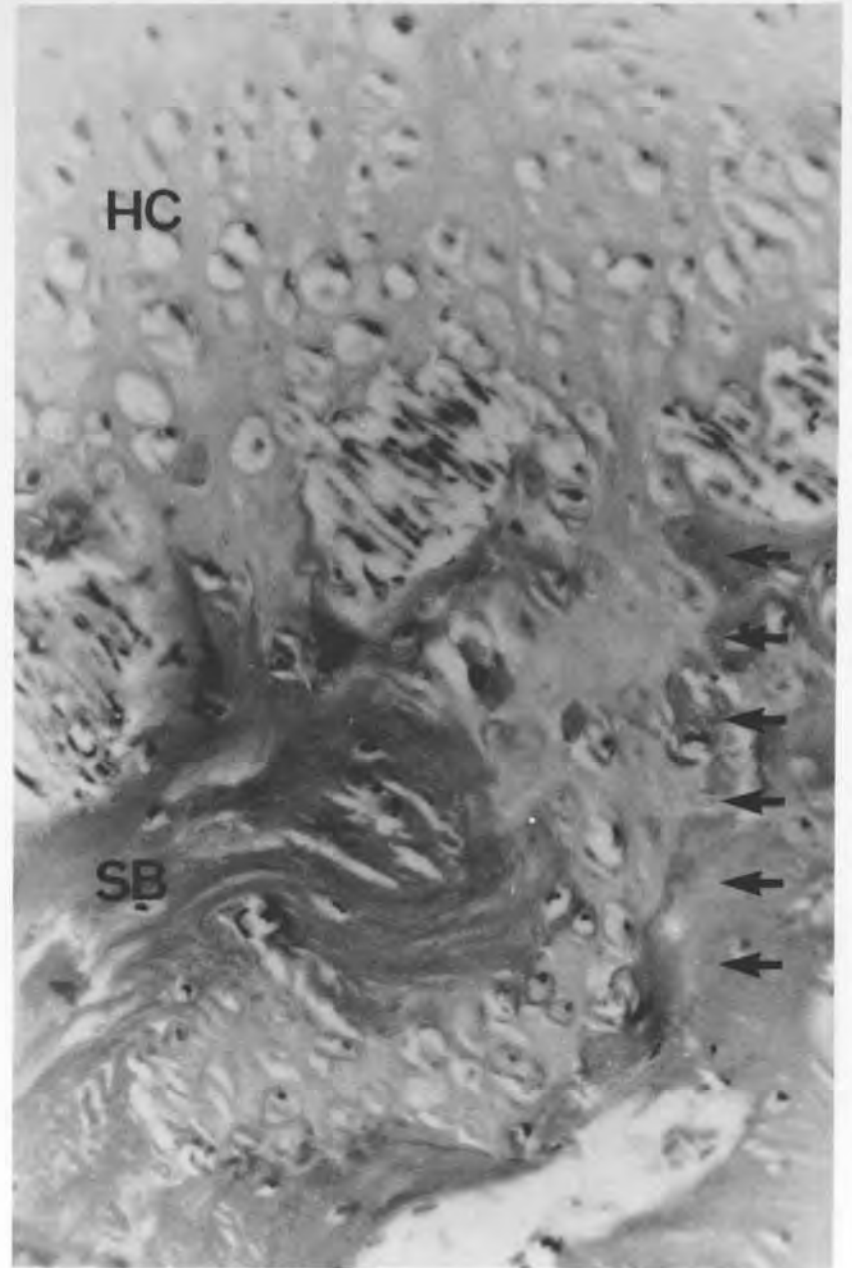


Fig. 3: Continuity of the hypertrophic cartilage (HC) and cartilage area in the subchondral bone (SB) (arrows) (H&E, original magnification $\times 200$).

Fig. 3: Continuité entre le cartilage hypertrophique (HC) et les territoires de cartilage au niveau de l'os subchondral (SB). (H.E. grossissement: $\times 200$).

Scanning electron microscopy

The articular surface exhibited undulating bundles of collagen fibers of different sizes. At higher magnifications, fibers showed a multidirectional distribution (Fig. 4). There were some clefts in the surface, with signs of dissociation of fibers (Fig. 5). Surrounding the clefts, there was loss of orientation of the collagen bundles, originating elevations protruding from the surface. Sparse globules, varying in diameter from 0.5 to 2 μm , were found over the surface, singly distributed or in clusters (Fig. 5).

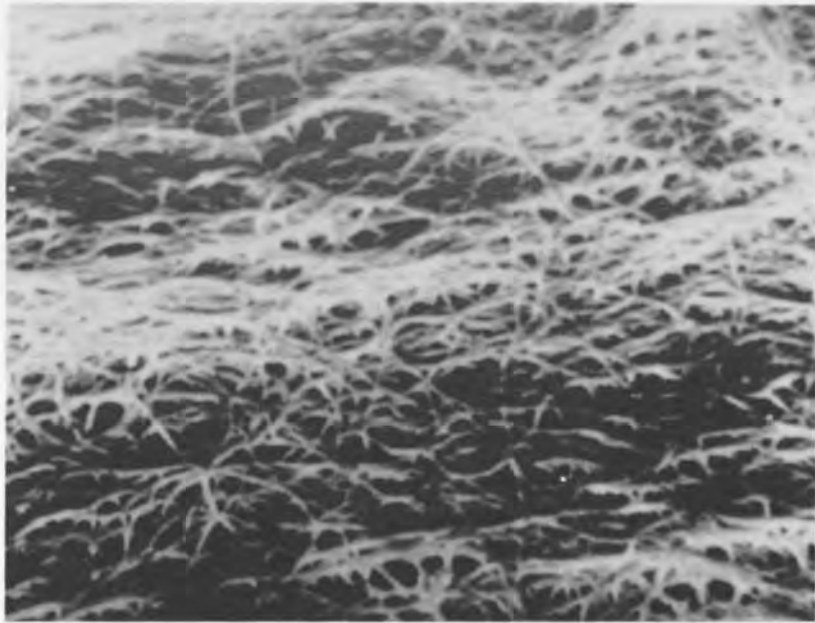


Fig. 4: Multidirectional distribution of collagen fibers of the articular surface (SEM, $\times 9000$).

Fig. 4: Distribution multidirectionnelle des fibres de collagène de la surface articulaire (MEB, $\times 9.000$).

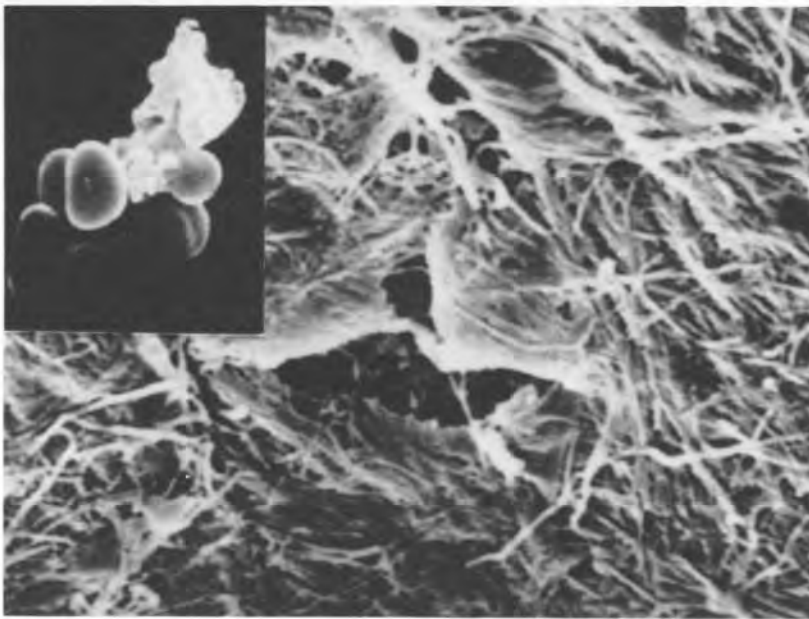


Fig. 5: Cleft in the articular surface (SEM, $\times 2000$). Top left, globules in cluster (SEM, $\times 2000$).

Fig. 5: Fissure dans la surface articulaire (MEB, $\times 2.000$). En haut à gauche: un amas de globules (MEB: $\times 2.000$).

Identification of different zones of the articular cartilage was possible but the interface between fibrocartilage and subchondral bone was difficult to distinguish. The articular zone was thick and composed of collagen fibers running parallel to the articular surface, with variable undulations. Giant

coiled fibers were frequent among collagen fibers (Fig. 6). The proliferative zone was composed of small, round cells and collagen fibrils (Fig. 7). Fibrocartilage presented collagen fibers in randomly oriented bundles.



Fig. 6: Image of articular cartilage in the sagittal plane. Giant coiled fibers in the articular surface are observed (SEM, $\times 200$).

Fig. 6: Cartilage articulaire en plan sagittal. On observe des fibres torsadées de grande taille dans la surface articulaire (MEB: $\times 200$).

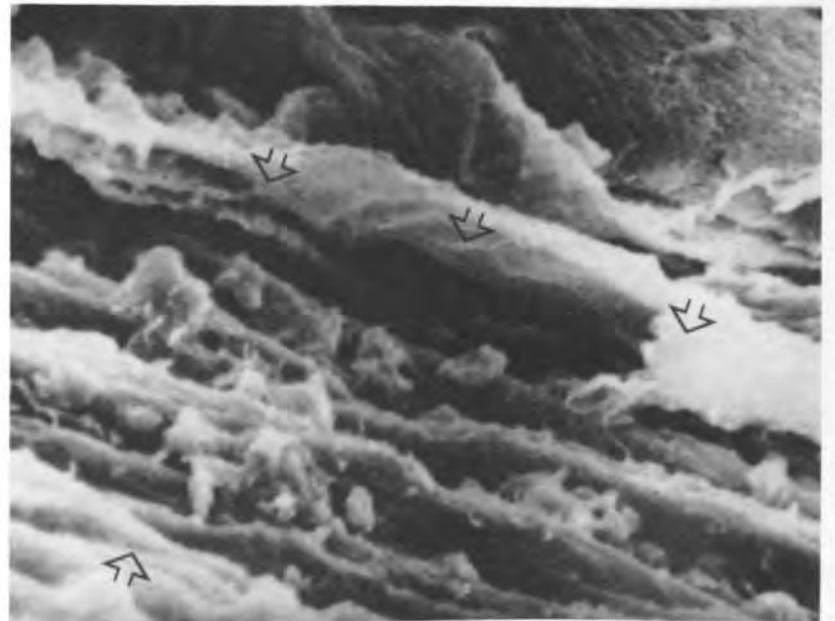


Fig. 7: Image of articular cartilage in the sagittal plane showing round cells in the proliferative zone (arrows) (SEM, $\times 800$).

Fig. 7: Cartilage articulaire en plan sagittal montrant des cellules rondes dans la zone proliférative (flèches) (MEB: $\times 800$).

DISCUSSION

Histological sections of both cases showed the presence of proliferative zone, hypertrophic chondrocytes, subchondral bone plate not closed, and cartilage rests in the cancellous bone. These data characterize activity of the articular cartilage layers (Öberg et al., 1962; Norman & Painter, 1980; Gray et al., 1990). The first case presented discontinuous articular surface, irregular proliferative zone, fibrocartilage with clustering of chondrocytes and a vertical cleft; the second case presented continuous and regular articular surface and proliferative zone, as well as thick hypertrophic cartilage zone. According to the age of onset the first case was an adult form of UCH and the second a juvenile form, and histopathological findings confirmed this classification (Slootweg & Müller, 1986).

Scanning electron microscopy revealed clefts on the articular surface, with surrounding elevations. These findings are characteristic of degenerative changes observed in articular surfaces with osteoarthritis (Redler & Zimny, 1970; Jagger & Whittaker, 1977; Wampler et al., 1980). The presence of clefts is related to disintegration of collagen fiber network due to osteoarthritic changes (de Bont et al., 1985b). These clefts are detectable only in ultrastructural studies, not being observed with light microscopy.

Another finding in the scanning electron microscopy of the articular surface was the occurrence of globules, varying 0.5-2 μ m in diameter. In the past, they were explained as synovial fluid droplets (Jagger & Whittaker, 1977).

Nowadays, they are related to degenerative changes observed in osteoarthritis of temporomandibular joint (TMJ). These globules are osmiophilic and considered as lipidic bodies, occurring as part of a degenerative process (de Bont et al., 1985b).

At the cutting surface, a significant finding of scanning electron microscopy was the occurrence of giant, coiled collagen fibers in the articular zone. Ultrastructural studies of degenerative changes in osteoarthritis at transmission electron microscope have shown alteration of collagen size and evidence of dissociation of both the collagen and its surrounding ground substance (Toller, 1977; Ghadially, 1983). Giant collagen fibers are considered a regressive change in articular cartilage (Ghadially, 1983). Another finding was the difficulty to distinguish the interface between fibrocartilage and subchondral bone, also related to osteoarthritis of TMJ (de Bont et al., 1985b).

Degenerative characteristics, i.e., discontinuous articular surface, clustering of chondrocytes in the fibrocartilage and clefts, similar to those present in osteoarthritis, have been related in the adult form of UCH (Slootweg & Müller, 1986). Otherwise, the finding of some degenerative characteristics, such as clefts and globules in the articular surface as well as alteration of collagen size, in the juvenile form, suggest that degenerative changes occur in UCH either in adult or juvenile forms.

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Correspondence to:

Dr. J.G.C. Luz,
Rua Duarte de Azevedo 284, s.22, 02036-021 São Paulo SP
(Brasil).