Early osteoarthritic changes of human femoral head cartilage
subsequent to femoro-acetabular impingement

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Summary

Objective: To use the surgical samples of patients with femoro-acetabular impingement due to a nonspherical head to analyze tissue morphology and early cartilage changes in a mechanical model of hip osteoarthritis (OA).

Design: An aberrant nonspherical shape of the femoral head has been assumed to cause an abutment conflict (impingement mechanism) of the hip with subsequent cartilage lesions of the acetabular rim and surface alterations of the nonspherical portion of the head. In this study, 22 samples of the nonspherical portions of the head have been obtained during hip surgery from young adults (mean 30.4 years, range 19–45 years) with an impingement conflict. The samples were first compared with tissue from the same area obtained from six age-matched deceased persons (control group) with normal hip morphology and second with cartilage from 14 older patients with advanced OA. All samples were characterized histologically and hyaline cartilage was graded according to the Mankin criteria. They were further subjected to examination on a molecular basis by immunohistology for cartilage oligomeric matrix protein (COMP), tenascin-C and a collagenase cleavage product (COL2-3/4Clong) and by in situ hybridization for collagen type I and collagen type II.

Results: All samples from the patient group revealed hyaline cartilage with degenerative signs. According to the Mankin criteria, the cartilage alterations were significantly different when compared with the control group (p=0.007) but were less distinct when compared with cartilage from patients with advanced OA (p=0.014). Positive staining and distribution pattern for COMP, tenascin-C and COL2-3/4Clong showed similarities between the samples from the impingement group and osteoarthritic cartilage but they were distinctly different when compared with healthy cartilage. Levels of collagen I and II transcripts were upregulated in 6 and 10, respectively, of the 14 samples with OA and in 9 and 12, respectively, of the 22 samples from the impingement group. None of the samples from the control group showed upregulation of Collagen I and II mRNA.

Conclusions: The aberrant nonspherical portion of the femoral head in young patients with an impingement conflict consists of hyaline cartilage which shows clear degenerative signs similar to the findings in osteoarthritic cartilage. The tissue alterations are distinctly different when compared with a control group, which substantiates an impingement conflict as an early mechanism for degeneration at the hip joint periphery.

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Key words: Hip, Impingement, Early osteoarthritis, In vivo.

Introduction

In recent studies an abutment conflict between the femoral head and the acetabular rim termed femoro-acetabular impingement has been postulated as cause of early degenerative cartilage lesions in young adults. As one of the causes, a morphological aberration of the femoral head presenting with a nonspherical shape has been proposed (Fig. 1). On vigorous flexion and internal rotation of the hip, the nonspherical portion is assumed to exert abnormal shear and compressive forces on the corresponding area of the acetabular cartilage comparable to a cam effect (Fig. 2). Patients typically present with inguinal pain that can be reproduced by an impingement test during clinical examination. Diagnosis can be confirmed by arthro-MRI, which reveals acetabular rim lesions and the aberrant morphology of the femoral head (Fig. 3). During open hip joint revision these cases nearly uniformly presented...
with delamination of the cartilage at the acetabular rim\(^1\) and redening, surface fraying and/or substantial surface irregularities of the nonspherical portion of the head [Fig. 4(a)]. Surgical resection of the nonspherical portion restores sphericity in this area. This will increase the range of impingement-free hip motion and should prevent or reduce ongoing mechanical damage to the acetabular cartilage. This procedure typically leads to a substantial decrease in symptoms\(^1\). The goal of this study consisted in morphological and histological analyses of the resected tissue from the nonspherical portion of the head.

The properties of the surgically removed tissue were first compared with the properties of tissue samples from the same area from age-matched fresh cadavers in order to exclude the possibility that these findings are incidental and represent an age-related ‘natural course’ of hip joint degeneration. The tissue samples from the impingement group were further compared with cartilage from patients with advanced osteoarthritic changes in order to evaluate the differences in the grade of cartilage degeneration between these two groups. The samples were examined for typical features of early osteoarthritis (OA) which include fibrilations of the superficial zone, later extending into the deeper layers, a net loss of proteoglycans and collagen type II and chondrocyte clustering\(^3\). OA is further characterized by an alteration of the distribution of a number of extracellular matrix molecules, such as fibronectin, tenascin-C and cartilage oligomeric matrix protein (COMP)\(^{16,20}\). Therefore, besides conventional histological staining methods, immunohistochemical staining for tenascin-C and COMP was used in the present study.

An integral feature of OA includes degradation of collagen II and disruption of its triple-helical ultrastructure. Although there is upregulation of collagen type II mRNA\(^4\), a net loss of type II collagen indicates that collagen degra-
Matrix metalloproteinases (MMPs) play a prominent role in the degradation of collagen type II as they are able to cleave its triple-helical domain. This cleavage site can be detected by COL2-3/4Clong, which was used in the present study as an antibody reactive to the carboxy terminal neoepitopes. Type II collagen is the main protein component of adult articular cartilage whereas collagen type I is normally absent in healthy cartilage except for a thin layer at the surface. For further characterization of the analyzed tissue, the gene expression and distribution of collagen II and I were evaluated on the mRNA level by in situ hybridization.

Material and methods

MATERIALS

(3-Aminopropyl)triethoxysilane, Safranin T and Fast Green FCF were purchased from Fluka (Steinheim, Germany). For immunohistochemistry (IHC), a polyclonal antibody against human COMP and a monoclonal antibody against human tenascin-C (clone B28) were provided by Dr M. Wong (M.E. Müller Institute for Biomechanics, University of Berne, Switzerland) and Dr R. Chiquet-Ehrismann (Friedrich Miescher Institute, Basel, Switzerland). An antibody against a collagenase cleavage product (COL2-3/4Clong) was obtained from HDM (Toronto, Canada). A secondary goat anti-rabbit IgG was purchased from ICN Biomedicals GmbH (Eschwege, Germany) as was a goat anti-mouse whole IgG. The mouse anti-peroxidase complex was obtained from Jackson Laboratories (West Grove, PA, USA). Chondroitinase ABC from Proteus vulgaris was purchased from Fluka and 3-amino-9-ethyl-carbazole from Sigma Chemical Co. (St. Louis, MO, USA). The in vitro transcription system, proteinase K and RNase T1 were purchased from Roche Diagnostics (Rotkreuz, Switzerland). GelBond film was obtained from FMC BioProducts (Rockland, ME, USA). Nitroblue tetrazolium (NBT) and 5-bromo-4-chloro-3-indoylphosphate (BCIP) were purchased from Sigma Chemical Co., Aquamount from BDH Laboratory Supplies (Poole, England) and all standard reagents from Merck (Darmstadt, Germany).

PATIENTS AND TISSUE SAMPLES

Cartilage samples from the hips were obtained during surgery from 22 patients with a mean age of 30.4 years (range 19–45 years). All patients presented with a typical history of femoro-acetabular impingement and a positive impingement test, i.e. inguinal pain provocation on flexion and internal rotation. Plain radiographs did not show osteophyte formations or loss of joint space width in any of the
hip joints, but revealed the typical morphology of a non-spherical head (Fig. 1). Acetabular rim lesions including alterations and tears of the labrum and the adjacent cartilage as well as the nonspherical femoral head shape were revealed on arthro-MRI in all of the patients. Treatment consisted in surgical resection of the nonspherical portion of the head, representing the impinging zone [Fig. 4(b)]. The resected portion was always confined anatomically to the anterior head–neck junction and always included a small layer of subchondral bone [Fig. 4(c)]. As control, samples from the same anatomical location were obtained in six age-matched (range 19–37 years) deceased persons. In addition, the samples were compared with advanced osteoarthritic cartilage, which was obtained from the central weightbearing zone of the femoral head from 14 older patients undergoing total hip joint replacement. The average age of these patients was 64 years (range 35–86 years). This clearly older patient group with advanced OA served as a ‘positive control’. Specimens from this group were obtained to gain cartilage in an ‘end-stage’ of hip joint degeneration. It was used to evaluate to which degree the pattern and grade of cartilage degeneration in the examined impingement group resembles the findings in patients with advanced OA. Cartilage samples from the OA group do not represent the areas with worst cartilage degeneration since they have been collected from sites with some residual hyaline cartilage, not from areas with complete bone exposure or those immediately next to it.

Immediately after removal, the tissue samples were fixed in 4% paraformaldehyde in phosphate buffered saline (PBS) for 24 h and decalcified in 15% EDTA pH 8.0. Complete decalcification was checked for by X-ray. Subsequently, the samples were dehydrated in graded ethanol, cleared in xylol and embedded in paraffin. The embedded tissues were cut into 3 µm sections and mounted on SuperFrost®Plus glass slides (Menzel-Gläser, Germany) for histological analysis or safranin-O fast green staining to estimate the content and distribution of proteoglycans. The morphology of the cartilage was graded according to the criteria of Mankin independently by a pathologist and a surgeon.

HISTOLOGICAL STAINING

All cartilage specimens were subjected to hematoxylin and eosin (H&E) for histological analysis or safranin-O fast green staining to estimate the content and distribution of proteoglycans. The morphology of the cartilage was graded according to the criteria of Mankin independently by a pathologist and a surgeon.

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For IHC sequential sections were chosen. Endogenous peroxidase was blocked by incubating the sections for 10 min in 0.5% H2O2 in methanol. Thereafter, the sections were washed in PBS (137 mM NaCl, 27 mM KCl, 83 mM Na2HPO4·12H2O, 5 mM KH2PO4; pH 7.4). To enhance permeability of the extracellular matrix, the sections were treated with chondroitinase ABC at 0.0125 U/50 µl in 0.1 M Tris-acetate buffer pH 7.2, for 90 min at 37°C. After washing, unspecific binding sites were blocked with 5% goat serum for 30 min. Subsequently, the sections were incubated (all incubations were performed for 1 h at room temperature) with one of the following antibodies: a rabbit polyclonal antibody against human COMP 1:200, a mouse monoclonal antibody against human tenascin-C, clone B28 1:100 and a mouse monoclonal antibody against the COOH terminus of the three-quarter fragment of collagen type II (COL2-3/4Clong) 1:50. After washing in Tris buffered saline (TBS) with 0.1% Tween 20, the sections treated with primary rabbit antibodies were incubated with peroxidase-conjugated goat anti-rabbit IgG 1:1000. Sections treated with primary mouse antibodies were incubated with goat anti-mouse whole IgG 1:500, and after washing with mouse anti-peroxidase complex 1:3000. After renewed washes, the IHC signals were revealed with 3-amin-9-ethylcarbazole as substrate.

IN SITU HYBRIDIZATION

In situ hybridizations were performed with slight modifications as described previously. After deparaffinization and rehybridization, the sections were digested with proteinase K (5 µg/ml) for 15 min at 37°C in 100 mM Tris–HCl (pH 8.0)/50 mM EDTA. Subsequently, the sections were refixed in 4% paraformaldehyde/PBS for 5 min at room temperature and washed 2×1 min in 2×standard saline-citrate buffered solution (SSC; 1× SSC=0.15 M NaCl, 0.015 M sodium citrate, pH 7.5). Acetylation was performed in 0.1 M triethanolamine (pH 8.0), containing 500 µl of acetic anhydride, for 10 min, upon which a second aliquot of 500 µl of acetic anhydride was added for another 10 min. The sections were prehybridized in 50% deionized formamide, 4× SSC, 2× Denhardt’s, and 250 µg/ml yeast tRNA for 2 h at 50°C and then hybridized in 20 µl of hybridization mix (50% deionized formamide, 4× SSC, 2× Denhardt’s, 1% dextran sulfate, 500 µg/ml yeast tRNA) containing 0.2 µl of DIG-labeled riboprobe. The sections were covered with FMC GelBond Film, placed in a humid box containing 50% deionized formamide in 2× SSC and incubated overnight at 50°C. After removal of the membranes, the sections were washed in 2× SSC for 30 min at room temperature, in 1 mM EDTA (pH 8.0) with 1 U/ml RNase T1 for 50 min at 37°C, then in 2× SSC for 2×10 min at 55°C, and finally in 0.2× SSC for 2×10 min at 55°C. After hybridization, the labeled riboprobes were detected by immunohistochemical analysis using an anti-DIG-antibody conjugated with alkaline phosphatase (Roche, IN, USA). Nonspecific binding sites were blocked with 1% blocking reagent (Roche Diagnostics, Rotkreuz, Switzerland) in maleate buffer (100 mM maleic acid, 150 mM NaCl, pH 7.5) for 30 min at room temperature. Anti-DIG ALP conjugate (200 µl), diluted 1:1000 in blocking solution containing 0.2% Tween 20, was overlaid on tissue sections and incubated for 2 h at room temperature. After 2×10 min washes in maleate buffer containing 0.2% Tween 20, the sections were equilibrated in ALP buffer (100 mM Tris–HCl (pH 9.5), 100 mM NaCl, 5 mM MgCl2) for 5 min at room temperature. Binding of the antibody was visualized by an enzyme specific reaction using BCIP and NBT system. Specificity of the hybridization experiments was assessed by hybridizing parallel sections with a sense (nonhybridizing) riboprobe for Col II. DIG-labeled riboprobes were obtained by linearizing pGEM-I plasmids containing type I rat collagen cDNA fragments of 600 bp from the C-terminal propeptide and type II human collagen cDNA fragments of 550 bp. After linearization of the plasmids in vitro transcription was performed using an in vitro transcription kit according to the recommendations of the manufacturer (Roche Diagnostics).
Histological changes observed in the three groups are illustrated in Fig. 5. According to the Mankin criteria, the grading of cartilage from the control group showed a mean Mankin grade of 1 (range 0–2) (Table I). There were no surface irregularities, no duplications of the tide-mark, representing the boundary between calcified and uncalcified cartilage and there was no chondrocyte cluster formation in five of the six samples [Fig. 5(a)]. One sample showed cluster formation with slight proteoglycan loss in its most lateral part of the hyaline cartilage.

The samples of the patient group with femoro-acetabular impingement showed a mean Mankin grade of 5.7 points (range 2–12) (Table I). The difference was statistically significant compared to the grading of the control group (Mann–Whitney U test; p=0.007). The hallmark observations in the patients were chondrocyte clustering and proteoglycan reduction, as seen in 17 of a total of 22 samples. Surface irregularities were found in 15 samples and six samples showed clefts to a different degree [Fig. 5(b,c)].

The osteoarthritic samples from patients undergoing joint replacement showed fissures and clefts and a severe reduction of safranin-O staining in the vast majority [Fig. 5(d,e)]. Grading according to the Mankin criteria showed a mean of 8.6 points (range 3–14). This difference was also statistically significant when compared to the patients with impingement (Mann–Whitney U test; p=0.0144).

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Tenascin-C staining of hyaline cartilage of the control group showed only a slight signal along the surface. No pericellular staining could be observed [Fig. 6(a)]. In cartilage from the impingement group an increase in signal was
also present along the surface of the cartilage. In addition, in all specimens from the impingement group there was tenascin-C positive staining in the pericellular and territorial regions in the upper middle zone and in the pericellular region in the lower middle zone [Fig. 6(b)]. In OA cartilage, samples staining for tenascin-C were found to be strongly increased along the surface and in the superficial zone [Fig. 6(c)]. In the majority of samples positive staining was also present in the pericellular and territorial regions of the middle zone. In samples with more advanced OA lesions this staining pattern could reach into the deep zone.

In healthy cartilage, the distribution of COMP was relatively uniform throughout the territorial and interterritorial matrix [Fig. 7(a)]. Cartilage samples from the impingement group in all samples showed an altered distribution with an intense staining for COMP in the pericellular and territorial regions in the superficial and middle zones [Fig. 7(b)]. In the deep zone interterritorial staining was observed. Chondrocyte clusters, which stained strongly for proteoglycans showed a negative staining for COMP within the territorial region. In OA cartilage staining for COMP was greatly increased in the superficial, fibrillated or fissured zone and there was territorial staining, which extended into the middle or deep zone [Fig. 7(c)]. In advanced cases of OA a complete loss of staining was observed in the deep zone. Chondrocyte clones that stained strongly for proteoglycans were negative for COMP.

A slightly positive staining of the pericellular region for COL2-3/4C long was found in the healthy control group [Fig. 8(a)]. In the impingement group 10 of 22 samples had a
positive staining for COL2-3/4C_long in the extracellular matrix in the superficial and middle zones [Fig. 8(b)]. These 10 cases also represented the samples with more advanced surface disruptions. All samples from the OA group showed a positive staining of the extracellular matrix for COL2-3/4C_long in the superficial and middle zones [Fig. 8(c)]. The staining pattern was similar to that in the impingement group. Control sections stained without the primary Ab or with preimmune rabbit serum were negative. Overall, there was a tendency for strong COL2-3/4C_long staining in cartilage with higher Mankin grades and with increased surface fraying, but this did not reach statistical significance.

**IN SITU HYBRIDIZATION**

In healthy hyaline cartilage mild to moderate signals for mRNA encoding collagen II were detected by *in situ* hybridization [Fig. 9(a)]. The positive signals were seen in all cartilage zones. In cartilage from the impingement group collagen II mRNA was upregulated in 12 of 22 cases. In six of the samples from the impingement group, the deep to middle zone was mainly affected [Fig. 9(b)]. Samples from OA cartilage presented with increased staining for collagen II in six of 14 samples. Especially cluster forming cells showed an upregulation of transcripts encoding collagen type II [Fig. 9(c)]. In three OA samples increased staining
for collagen II mRNA was observed in the deep to middle zone only. Samples from the healthy control group did not show a signal for collagen $\alpha_1$($I$) mRNA above background [Fig. 10(a)]. Samples from the impingement group had a positive signal for $\alpha_1$($I$) mRNA in nine of 22 cases mainly in the deep zone [Fig. 10(b)]. Five of these nine cases presented with a high signal intensity. In osteoarthritic cartilage, collagen $\alpha_1$($I$) mRNA was detected in seven of the 14 samples within the degenerated hyaline cartilage, whereas four osteoarthritic samples showed a very strong signal in the deep zone [Fig. 10(c)]. In most sections cells in bone were positive for transcripts of collagen $\alpha_1$($I$) which served as a positive control. No statistically significant correlation between increased collagen I or II mRNA levels and the Mankin grades could be demonstrated. In summary, there was a tendency for a higher number of samples with increased signals for collagen I mRNA in the OA group compared with higher numbers with increased signals for collagen II mRNA in the impingement group. These differences did not reach statistical significance either.

**Discussion**

The aim of this study was to investigate histological and molecular alterations in tissue samples from abnormal nonspherical portions of the femoral head in a distinct patient group. In these young patients, the abnormally shaped femoral head is considered to cause intraarticular lesions by a femoro-acetabular impingement mechanism. Thereby, one may assume that the nonspherical portion of the head is subject to abnormal shear and compressive forces. All the examined specimens revealed hyaline cartilage with distinct signs of degeneration, which were clearly different when compared with cartilage from the same area in a healthy control group. These differences were evident by grading according to the Mankin criteria as well as in the expression and altered distribution of OA-associated proteins or cleavage products (tenascin-C, COMP, COL2-3/4C<sub>long</sub>) and mRNA transcripts for collagen $\alpha_1$($II$) and collagen $\alpha_1$($I$). The samples from the presented patient group showed a similar pattern of cartilage degeneration as seen in samples from femoral heads with advanced OA. Although the hyaline cartilage samples from the OA group may not represent the areas with strongest degeneration, which would be characterized by bare bone or fibrocartilage formation, the mean Mankin score was still statistically significantly higher compared with the findings in the impingement group. Thus, according to the Mankin score the cartilage alterations in the impingement group seem to represent a moderately earlier stage of degeneration than typically seen in patients undergoing THA.

Changes found by IHC and *in situ* hybridization on the other hand did not reveal significant differences between the impingement and the OA group. This may be due to the variant grade of cartilage degeneration in both groups and/or to a limited number of samples. It may also indicate that in the examined range of cartilage degeneration the alterations on an enzymatic and immunohistochemical level may follow a similar pathway and are less dependent on the underlying pathomechanism or the histological grade of degeneration.

The study of cartilage alterations on different levels should help to compensate for restrictions in interpretations of results obtained by a single method. It remains unclear whether the increased positive staining for tenascin-C and COMP in cartilage from patients with hip joint impingement or advanced OA is related to an increased synthesis of the specific protein or to an increased accessibility. A purely enhanced accessibility seems unlikely since the areas of proteoglycan loss generally are not overlapping with areas with increased staining for these two glycoproteins. The method does not allow a differentiation whether a potentially increased synthesis is due to an upregulation in chondrocytes or synovial cells. A positive staining for collagen II cleavage products with the use of COL2-3/4C<sub>long</sub> antibody reveals a breakdown of the major matrix collagen attributed to early degeneration in OA.
In situ hybridization for collagen I and II mRNA helps to detect an altered gene expression and upregulation within the chondrocytes, which can be interpreted as an attempt for cartilage repair.9

COMP has been shown to be synthesized and secreted by synovial cells and by chondrocytes. In cartilage specimens from the impingement group an increased signal for COMP was seen at the surface and in the territorial region of the chondrocytes from the superficial to middle zone, as typically reported for OA. Since synovial cell production of COMP is known to be increased during degeneration or abnormal loading it also remains unclear for COMP whether an increase in signal of the superficial and middle zones is due to increased uptake from the synovial fluid or increased synthesis within chondrocytes. Upregulation of COMP under cyclic loading in bovine cartilage rather supports an increase of synthesis within the chondrocytes. This is further supported by the fact that in advanced cases of OA (Mankin grade ≥9) of the present series, where chondrocytes appeared dead, staining for COMP was absent in the interterritorial region of the deep zone as opposed to cases with less severe OA (Mankin grade <9).

Tenascin-C staining was increased within the superficial to middle zone in cartilage from the impingement group and in osteoarthritic cartilage, which was in contrast to the staining in healthy cartilage. This is consistent with earlier findings in OA and with a previously demonstrated increased tenascin-C production within chondrocytes. As tenascin-C counteracts the fibronectin induced cartilage resorption in OA, a potential explanation might be that the increased expression could represent an attempted repair process starting within the superficial zone. Tenascin-C is expressed in various tissues bearing high tensile stress. Thus, another explanation for the increased pericellular staining in these zones theoretically might be increased shear stresses due to the impingement mechanism in this specific patient group.

The collagenase cleavage product consisting of the COOH terminus of the three-quarter fragment of collagen type II was found in 10 of the 22 cases with impingement and in all of the OA cartilage samples, whereas none of the healthy cartilage samples stained for COL2-3/4Clong. The observed distribution of the positive staining in the superficial zone is consistent with the pattern seen with a polyclonal Ab directed against the same antigen in an OA model for rats. Several collagenases have been shown to play an important role in OA by collagen II denaturation thereby exposing a selective cleavage site. Positive antibody staining for this selective cleavage site in nearly half of the presented patient group therefore indicates an osteoarthritic process in hips with femoro-acetabular impingement.

An upregulation of collagen α1(II) mRNA was observed in 12 of 22 patients with impingement and in six of 14 patients with advanced OA. Upregulation was predominantly present in cluster forming chondrocytes. In samples with an earlier stage of degeneration (lower Mankin grades) up-regulation of α1(II) mRNA typically was seen in the middle and deep zones and as a trend, extended to the superficial zone with more advanced OA, represented by higher Mankin grades in the present series. An increase of collagen α1(II) mRNA especially in the deep and middle zones has been associated with an attempt of active repair in OA by several authors. This is reinforced by a similar distribution described for C-propeptide of type II procollagen in OA. Whether this always is correlated with a state of cartilage degeneration or may also represent a reactive process to abnormal loading as it may be assumed in patients with impingement remains to be elucidated.

For collagen type I synthesis and its occurrence in OA, literature presents with divergent data. Cartilage explants from OA joints in culture have shown to synthesize collagen type I. Collagen I was also detected by immunohistochemical analysis typically around chondrocyte clusters in OA in the superficial and middle zones. On the other hand, Aigner et al. could not detect signals for mRNA encoding Col α1(I) in osteoarthritic cartilage by in situ hybridization. In the present study, no transcripts encoding Col α1(I) could be detected in the samples from healthy cartilage. Although the cartilage from the impingement group showed significantly lower Mankin stages of degeneration than cartilage from the OA group, Col α1(I) mRNA levels were increased in both groups. Increased levels of Col α1(I) mRNA were found mainly in the deep zone in nine of 22 cases with impingement and in seven of 14 cases with advanced OA. One may hypothesize that the cartilage from the impingement group is subjected to abnormally high stresses which may cause a change in the phenotypic gene expression of chondrocytes. A shift in the patterns of collagen synthesis has been observed under experimental conditions. Thereby, OA of the knee joint in rabbits was induced by medial meniscectomy and transection of the medial and anterior cruciate ligaments and lead to positive immunohistochemical staining for type I collagen. Upregulation of α1(I) mRNA might be due to a dedifferentiation of chondrocytes in OA or, as Focht and Adams assumed, the transcription of type I collagen genes may never be inactivated but the expression of the protein may be regulated exclusively at the translational level.

In conclusion, the cartilage samples from the non-spherical portion of the femoral head in young patients with femoro-acetabular impingement showed local degenerative changes consistent with findings in OA. These cartilage alterations are in contrast to the findings in a control group and thus strongly suggest abnormal loading of this portion of the femoral head. The widely accepted theory for the development of OA in the hip is based on mechanical overload of the cartilage in the central weight-bearing area. In contrast to this initial development of OA of the hip in the central weightbearing area the findings in the presented patient group showed distinct signs of cartilage degeneration at the hip joint periphery. Both, the alterations at the acetabular rim as well as at the head periphery can be visualized during surgery and are in clear contrast to normal appearing cartilage in the central weightbearing areas [Fig. 4(a)]. This supports the femoro-acetabular impingement mechanism as a cause for early OA in this selected patient group and it seems likely that the lesions at the joint periphery may further trigger OA and lead to generalized OA of the hip.

Acknowledgements
We wish to thank Jeanette Portenier for technical assistance and Dr Marcy Wong who kindly provided the COMP antibody.
References


