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Invited Interesting Image

대퇴골 관절돌기 비분리성 박리 뼈연골염의 ^{99m}Tc-HDP 핀홀 뼈소캔 소견 1예보고: X선, CT 및 MRI 소견과의 비교검토

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^{99m}Tc-HDP Pinhole Bone Scan Features of Undetached Osteochondritis Dissecans of the Femoral Condyle: Report of a Case with Radiography, CT, and MRI Correlation

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Introduction

Osteochondritis dissecans (OCD) is a rare disorder of bone and cartilage of the femoral condyle especially after the second or third decade of life.[1,2] Congenital factors,[3,4] trauma,[5] inflammation, and others have been proposed as possible etiologies and physical injury plays a key role. According to Barrie's hypothesis[6] OCD may be initiated by arrest of bone growth and subchondral osteosclerosis followed by either cartilage hypertrophy with calcification or enfolding with osteochondral bridging. Diagnosis can be made in most instances using magnetic resonance imaging (MRI) or computed tomography (CT) or invasive arthroscopy. As to usefulness of conventional radiography (CR) opinions diverge as some held it to be of limited value[7,8] while others valuable.[9] The controversy seems to be due to semantic confusion of OCD from osteonecrosis (ON) which are different entities. This report will describe a case of undetached OCD occurred in the medial femoral condyle in a middle-aged female. It was free of symptom and incidentally discovered on ^{99m}Tc-HDP pinhole scan performed for patellar injury. Pinhole scan findings of OCD are correlated to those of CR, CT, and MRI. An electronic search of literature failed to reveal earlier publication of bone scan features of undetached OCD.

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Case Report

The patient was a 58-year-old obese female admitted because of multiple contusions and lacerations sustained in a motor vehicle collision. The main injury was bleeding skin laceration of the right knee including the patella. Admission CR of the right knee was first thought to be negative except for degenerated patella but a second look revealed barely discernible bony derangement in the undersurface of the lateral femoral condyle (Fig. 1A). In contrast CT displayed a well-defined, ovoid, radiolucent lesion with a thin sclerotic wall in the subcortical bone of the condyle (Fig. 1B) and pinhole scan showed an ovoid lesion with very intense ^{99m}Tc-HDP uptake (Fig. 1C). Of interest the lesion was surrounded by a thin halo of lower tracer uptake (Fig. 1C, 2A). There was prominent tracer uptake in the painful patella denoting contusion and fractures. MRI confirmed the presence of a lesion at the condylar undersurface with mixed signal intensities on T1

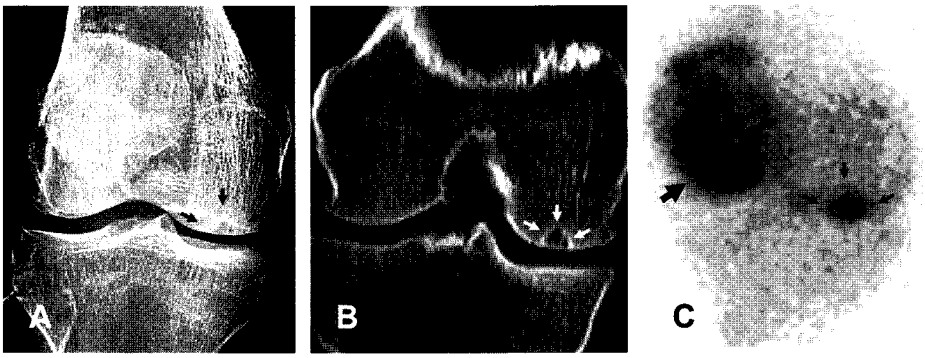

Figure 1. A. Anteroposterior radiograph of right knee shows small ovoid area of barely discernible architectural change of cancellous bone at medical femoral condylar undersurface (arrows). Lesion was retroscopically recognized only after noting pathology on pinhole scan and CT. B. Coronal CT shows small well-demarcated ovoid radiolucent lesion with thin sclerotic margin (arrows). C. Anterior pinhole scan shows ovoid focus with prominent tracer uptake surrounded by halo of lower tracer uptake (small arrows). Large irregular area of intense tracer uptake in patella is due to contusion and fractures (large arrow).

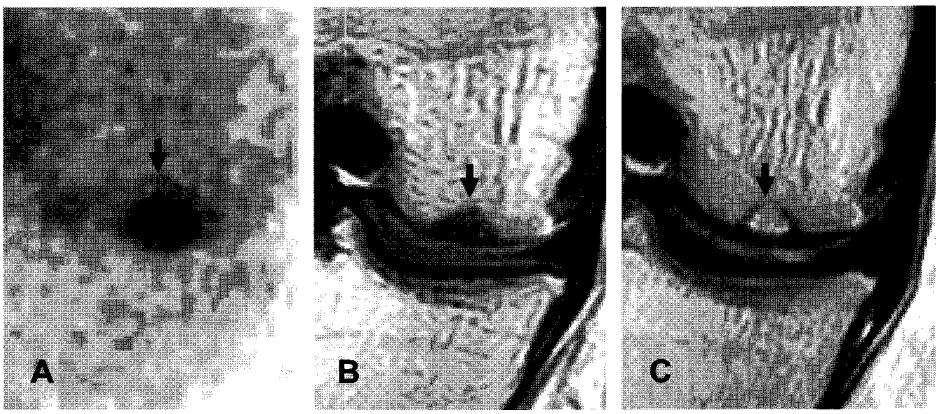

Figure 2. A. Close-up anterior pinhole scan of right medial femoral condylar undersurface clearly shows halo of sclerosis to surround undetached OCD (arrow). Tracer uptake in halo is lower than that of OCD denoting that OCD is not detached and connected by sclerosis (arrow). B. Close-up T1-weighted MR image (TR/TE = 480/29) shows both fragment and halo to consist of mixed signal intensities (arrow). C. T2-weighted MR image (TR/TE = 3800/92) shows bright signal intensity in OCD and low signal halo in sclerosis (arrow).

weighted image and bright signal intensity on T2 weighted image (Fig. 2B, 2C). Thus, the diagnosis of undetached OCD was suspected on CR, strongly indicated by pinhole scan, and substantiated by CT and MRI.

Discussion

OCD is also referred to as transchondral fracture. As to etiologies genetic factor or trait,[3,4] trauma,[5] inflammation, and others have been suggested but none has been proven to be definitive. Of these trauma has been shown to be causative in over 50% of cases[5] and inflammation has been discarded.[2] According to Barrie's hypothesis[6] OCD may be initiated by the arrest of bone growth followed by subchondral sclerosis by either cartilage hypertrophy with calcification or enfolding with osteochondral bridging. The fragment in OCD may assume one of four forms including *in situ*, partially detached, completely detached, or liberated within joint space.[2] To those one might add the "undetached" type as seen in the current case.

Pathologically, OCD differs from ON in that the fragment in the former condition comes off from a normal vascular bony bed while that in the latter separates from an avascular bony bed.[10] Indeed, bone fragment in ON is devascularized but that in OCD maintains vascularity until

it will have been completely detached. Hence, the fragment in ON cannot accumulate tracer presenting as a "cold" defect on bone scan while that in undetached or partially detached OCD accumulates tracer creating a "hot" lesion as in the current case. It was of interest that OCD with higher tracer uptake in our case was encircled by a small halo of lower uptake of presumably sclerosis that was not detached from the host bone (Fig. 2A).

As to the diagnostic approach CR has been described to be of little value in OCD by some authors[7,8] while others held it useful.[9] The controversy seems to be the result of semantic confusion of OCD and ON. It is understandable that *in situ* or undetached OCD is difficult to diagnose on CR as in our case (Fig. 1A) but once detached diagnosis is not so difficult. In contrast CT and MRI have been shown to be sensitive and specific as shown in Figures 1B, 2B, and 2C. Pinhole magnification bone scan appears to be very helpful in distinguishing dead fragment from alive. It can also provide information about the presence or absence of bony connection between the fragment and bony bed by presenting the "halo" of lower tracer uptake (Fig. 2A). MRI clearly depicted fragment and halo. The fragment manifested mixed signal intensities and bright signal intensity on T1 weighted (Fig. 2B) and T2 weighted images (Fig. 2C), respectively and the halo showed low signal intensity on both T1 and T2 images (Fig. 2B, 2C).

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