Idiopathic Osteonecrosis of the Patella: An Unusual Cause of Pain in the Knee

A CASE REPORT*

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We are reporting the case of a patient who had idiopathic osteonecrosis of the patella. To our knowledge, this is the first such report.

Case Report

A sixteen-year-old white girl had a history of pain in the right knee for four months, especially in the medial joint line, and occasional episodes of giving-way during sports activities. She could recall no trauma to the knee. Examination of the knee was unremarkable except for slight tenderness over the medial joint line. There was no effusion, and the McMurray and Lachman tests were normal.

Radiographs showed a poorly defined ovoid defect with a sclerotic margin in the superolateral part of the right patella; this was apparently asymptomatic and was thought to be benign. Radiographs of the left knee were normal. An arthrogram showed a probable tear of the medial meniscus of the right knee.

Two months later, arthroscopy revealed a small parrot-beak tear of the lateral meniscus and a normal medial meniscus. The articular cartilage, including that of the patella, appeared to be entirely normal. The torn part of the meniscus was shaved to form a smooth rim.

Two months after arthroscopy, the patient began to complain of new pain over the superolateral aspect of the patella of the right knee. The pain occasionally radiated around the entire knee and frequently awakened her at night. She had no fever or chills.

On physical examination, the pain was reproducible on palpation of the superolateral part of the patella. There was no effusion or increased warmth of the knee. The knee had a full range of motion, with mild pain but no crepitus; the pain seemed to be confined to the patella or patellofemoral mechanism. The circumference of the thigh at a point ten centimeters proximal to the proximal pole of the patella was equal to that of the contralateral thigh.

The results of laboratory studies, including a complete blood count and erythrocyte sedimentation rate, were normal.

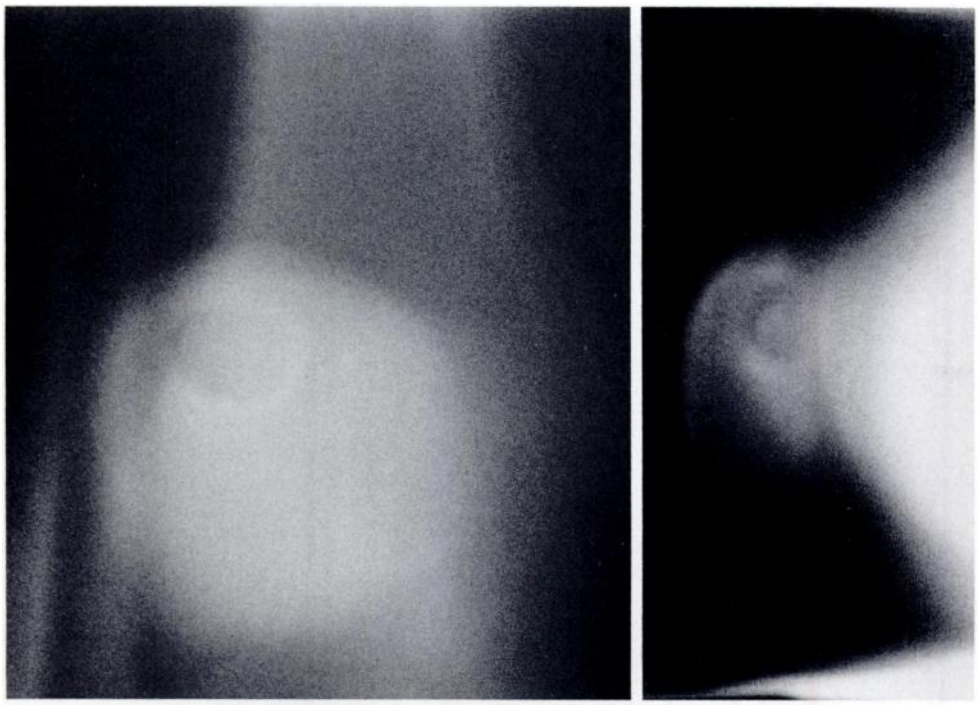

FIG. 1-A

FIG. 1-B

Fig. 1-A: Conventional anteroposterior tomogram of the right patella. A poorly demarcated area of radiolucency is seen in the superolateral region.
Fig. 1-B: Conventional lateral tomogram of the right patella. (No involvement of the articular surface or surrounding cortical bone was seen at the operation.)

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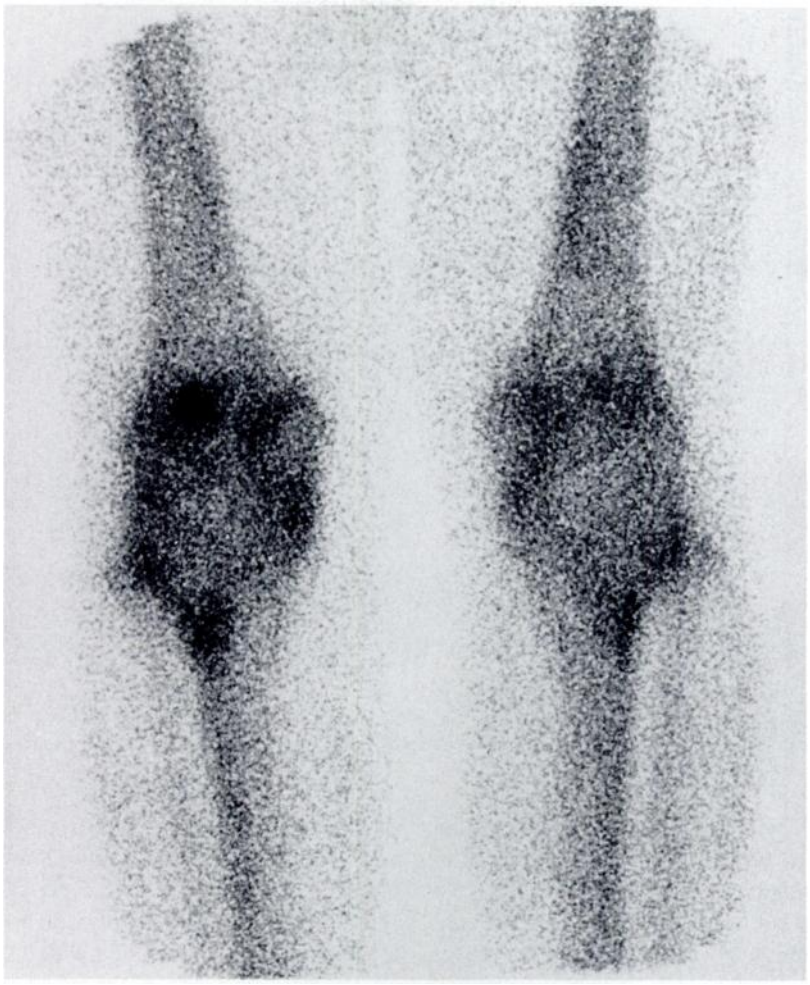

FIG. 2

99m-technetium-diphosphonate scintiscan of the knees. Increased uptake can be seen in the superolateral region of the right patella.

Non-steroidal anti-inflammatory medications relieved the pain somewhat initially, but soon became ineffective.

Conventional tomograms of the right patella revealed a poorly demarcated radiolucent area (Figs. 1-A and 1-B). A radionuclide bone scan showed increased uptake in the superolateral region of the patella (Fig. 2).

At the operation, the lesion was approached anteriorly, with the dissection remaining extra-articular except for a two-centimeter lateral parapatellar arthrotomy for inspection of the articular cartilage. Grossly, the lesion appeared to be avascular, with a slightly hyperemic, poorly defined border. The lesion was outlined with four Keith needles, and intraoperative radiographs were made to verify its position. The lesion was then excised with an osteotome and curet, with care being taken to preserve the articular cartilage. Intraoperative radiographs showed that the lesion had been completely excised. The defect, which was approximately one by one centimeter, was then filled with autogenous cancellous bone from the iliac crest, and the periosteum was closed over the graft. The articular cartilage remained intact, with a normal, pearly-white appearance.

The histological appearance of the specimen was consistent with osteonecrosis (Figs. 3-A and 3-B). Peripherally, bone had been largely replaced by well vascularized fibrous tissue. Centrally, the osseous trabeculae were necrotic, with empty osteocyte lacunae and loss of normal architecture.

After excision of the lesion, the symptoms subsided. The patient remained asymptomatic at the most recent follow-up examination, thirty months later.

Discussion

Osteonecrosis of the patella has been reported after trauma[24], use of steroids[28], and total knee arthroplasty[25]. It is considered to be rare; we found fewer than fifty cases in the literature. To our knowledge, this is the first reported case of idiopathic osteonecrosis of the patella.

The cause of idiopathic osteonecrosis is unknown, but the lesion appears to be due to multiple factors rather than a single cause. Many conditions and diseases have been associated with osteonecrosis, including systemic administration of steroids[4,9], alcoholism[15,19], sickle-cell disease[26], gout[16], rheumatological disorders[21], caisson disease[14,17], trauma[16], acute pancreatitis[10], and familial hyperlipidemia[16].

Many pathogenic mechanisms have been experimentally demonstrated. Patients who are treated with corticosteroids, as well as those who abuse alcohol, have an increase in the size of fat cells within bone-marrow cavities, and it has been postulated that this raises intraosseous pressure and diminishes perfusion[5,27]. Fatty emboli have been seen in subchondral bone after administration of steroids both clinically and experimentally[6,18]. Intraosseous microemboli localize in subchondral regions after intravenous

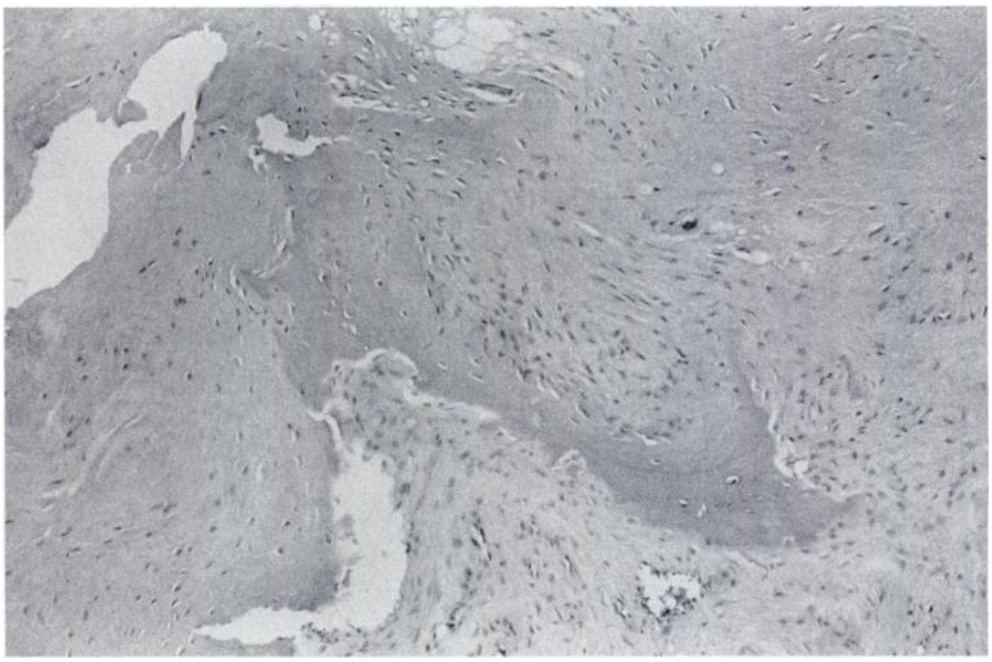

FIG. 3-A

Figs. 3-A and 3-B: Histological appearance of the patellar lesion.
Fig. 3-A: Peripherally, well vascularized fibrous tissue has begun to replace necrotic bone in the process of revascularization (hematoxylin and eosin, × 100).

injection experimentally[11] and could be the cause of osteonecrosis in patients who have gout (urate crystals), sickle-cell anemia (clumps of sickle cells), caisson disease (nitrogen bubbles), or another hormonal or hematological condition that increases levels of serum lipids or causes abnormal coagulation of blood. An "accumulative cell stress hypothesis" has also been proposed for osteonecrosis associated with diseases in which bone cells are purported to be weakened by systemic disease and are especially susceptible to these mechanisms or to a direct cellular toxic effect[20].

In the reports on patients who had patellar osteonecrosis after trauma or in association with the use of corticosteroids, all occurrences were in the proximal pole. With trauma, there was either a transverse fracture, with the edges widely separated before operative repair, or severe prepatellar soft-

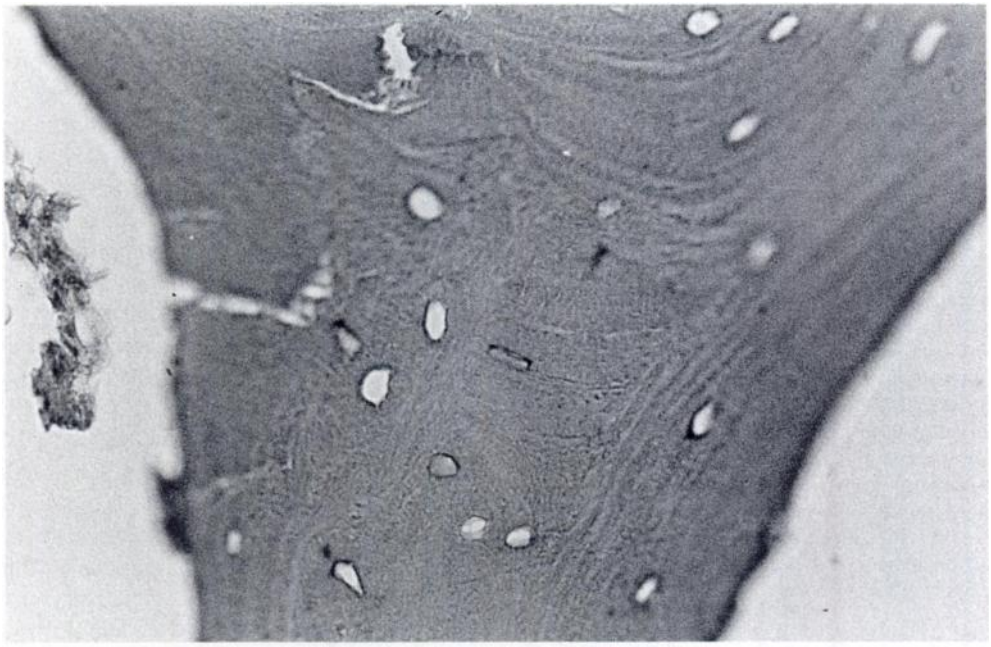

FIG. 3-B

Centrally, the osseous trabeculae are necrotic. Empty osteocyte lacunae and loss of normal architecture are seen (hematoxylin and eosin, × 400).

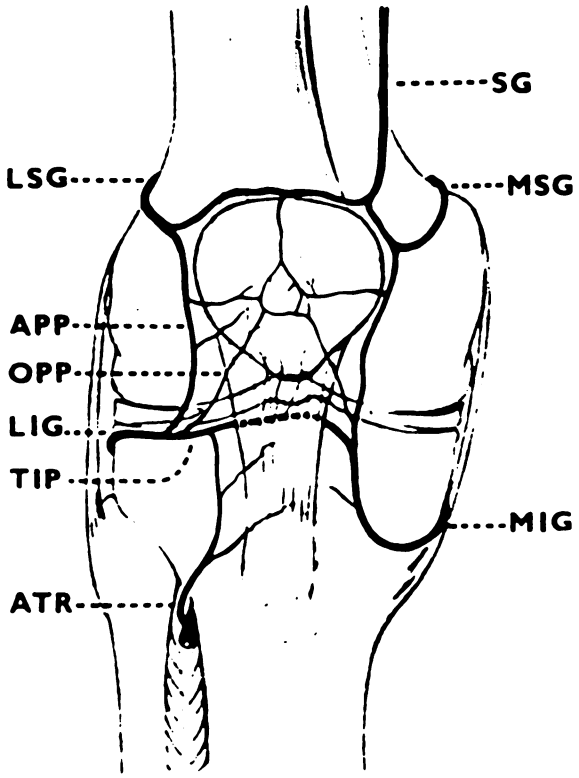

FIG. 4

The blood supply to the patella[24]. An anastomotic ring of vessels enters the anterior surface obliquely and provides the main blood supply to the proximal part of the patella. Apical or polar arteries enter the inferior part of the patella and run superiorly, supplying the inferior portion of the patella. SG = articular branch of the descending genicular artery (formerly called the supreme genicular artery), MSG = medial superior genicular artery, MIG = medial inferior genicular artery, LSG = lateral superior genicular artery, APP = ascending parapatellar artery, OPP = oblique prepatellar artery, LIG = lateral inferior genicular artery, TIP = transverse infrapatellar artery, and ATR = anterior tibial recurrent artery. (Reprinted, with permission, from: Scapinelli, Raffaele: Blood Supply of the Human Patella. Its Relation to Ischaemic Necrosis after Fracture. J. Bone and Joint Surg., 49-B(3): 564, 1967.)

tissue damage[24]. All patients in whom patellar osteonecrosis developed after a total knee arthroplasty had had a lateral release procedure[25], which probably interrupted the blood supply to the patella.

A study of the blood supply to the patella suggests why the proximal pole may be prone to the development of osteonecrosis. There are two main sources of blood to the patella: an anastomotic ring of blood vessels (Fig. 4) that enters the anterior surface obliquely and provides the main blood supply to the proximal part of the patella[2,24,28], and the apical or polar arteries that enter the patella inferiorly from deep to the patellar ligament and run superiorly to supply the inferior half of the patella[2,24]. Secondary sources of blood supply to the patella are arteries that enter from the interior of the quadriceps tendon, the synovial tissues, and the medial and lateral retinacula[2].

The diagnosis of osteonecrosis is usually based on radiographic and histological findings. Radiographically, osteonecrosis in its early stages appears as a sclerotic region in bone, and areas become radiolucent as resorption occurs. Radioisotopic bone scans are positive because of increased uptake of bone-seeking isotopes in the areas of microfractures and healing[7,12,13]. Histologically, the osseous trabeculae are seen to have empty lacunae. The bone marrow can be filled with eosinophilic amorphous and cellular material. The necrotic region is surrounded by fibrovascular granulation tissue and reparative areas of new-bone formation[1,13].

The delay in the onset of well localized pain in this patient is not unusual for osteonecrosis. In some patients, pain (which seems to develop at the interface between normal and necrotic bone, for some reason) may take as long as eighteen months to develop and localize[3]. Pain may be present at night, and there may be tenderness on direct pressure over a lesion[23]. The natural course of untreated painful osteonecrosis of the patella is unknown.

The cause of osteonecrosis in this patient remains obscure. One might speculate that it was related to the placement of the inflow catheter at the time of arthroscopy, but a medial inflow portal was used and the necrotic lesion had been present preoperatively. It has been speculated that microfractures in subchondral bone and cartilage can raise the intraosseous pressure as joint fluid is forced into the marrow space[23], but the articular cartilage was found to be intact. There was no history of trauma that would have reduced the supply of blood, nor was there any evidence of lateral patellar subluxation, which could have caused an increase in shear stress on the lateral portion of the patella. The region of osteonecrosis in this patient was probably not routinely subjected to abnormal repetitive stress, since that occurs in the part of the patella that does not contact the femoral condyles until the knee is in almost 90 degrees of flexion[8]. There also was no evidence of an open secondary ossification center or bipartite patella to suggest an abnormal blood supply in this region[22], and radiographically there was no evidence of a bipartite patella in either knee.

The diagnosis of idiopathic osteonecrosis of the patella was made with radiographs, a radioisotopic bone scan, conventional tomograms, and histological examination after excision. Idiopathic osteonecrosis should be considered as a possible cause of pain in the knee.

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