OSTEOCHONDROSIS OF THE SUPERIOR POLE OF THE PATELLA: TWO CASES WITH HISTOLOGIC CORRELATION

Wakenda Tyler,[1] BA and Edward F. McCarthy,[2] MD

ABSTRACT

Two cases of osteochondrosis of the superior pole of the patella are reported with histologic findings. Both patients were young girls; one had mild cerebral palsy. Sixteen cases of this disorder have been documented but without histologic study. The histologic features of these two cases showed osteonecrosis with reparative changes. These findings support that this entity is similar to other osteochondroses of the quadriceps mechanism: Osgood-Schlatter disease and Sinding-Larsen-Johansson disease.

INTRODUCTION

The osteochondroses are a heterogeneous group of injuries to the epiphyses and apophyses of children or adolescents.[6] Many of these injuries result in osteonecrosis in the avulsed fragment. Radiographically, they are characterized by bone fragmentation and sclerosis.[6] There are at least twelve eponymic syndromes among this group of illnesses. The spectrum of sites ranges from large epiphyses like the femoral head (Legg-Calve-Perthes' disease) to the small metatarsal head (Freiberg's infraction). Among the osteochondroses are two well-known syndromes associated with the quadriceps mechanism: 1) Osgood-Schlatter disease, an avulsion of the tibial tuberosity, and 2) Sinding-Larsen-Johansson disease, a chronic avulsion injury of the lower pole of the patella. A less well-described injury to the patella is chronic avulsion and fragmentation of the superior pole. We present two cases of female children with osteochondrosis of the proximal pole of the patella, review the literature, and document the histological features for the first time.

CASE REPORTS

Case 1

J. T. is an eleven year-old girl who initially presented with a right knee effusion, right knee pain, and mild scoliosis of the thoracic spine. She reported no prior history of trauma to her knee. Physical exam revealed a mild right knee effusion, but there were no other remarkable findings. She received no further work-up for her knee at that time. Two years later, she was still having pain. In addition, there was increased bulk of the right leg musculature compared to the left side, and there was slight worsening of her scoliosis. Physical exam at this time revealed hypertrophy of the quadriceps muscle group and calf muscles with spasticity and a five-degree flexion contracture on the right side. She was diagnosed with mild cerebral palsy. The right patella was slightly larger than the left, and the right leg was 1/2 cm longer. Radiographs revealed fragmentation of the proximal pole of the patella (Figure 1). The fragments were excised and the quadriceps reattached. The histological findings revealed extensive osteonecrosis with florid reparative changes consistent with osteochondrosis (Figure 2).

Case 2

L. R. is a seven year-old girl who initially presented with mild right-sided knee pain without a history of trauma. On physical exam, her right patella was noted to be slightly larger than her left, but there were no other findings. Radiograph taken at this time revealed no abnormalities, and she was thought to have juvenile rheumatoid arthritis. A year later she was re-evaluated after failure of medical treatment. Physical exam revealed a markedly enlarged right patella compared to the left side. The radiographs showed an enlarged patella with superior pole radiodensities and patellar irregularity at the superior pole (Figure 3). The proximal two thirds of the patella was excised, and histologic study documented osteonecrosis and reparative bone (Figure 4).

[1]Johns Hopkins School of Medicine
Baltimore, MD
[2]Johns Hopkins Medical Institution
Department of Pathology & Orthopedic Surgery
Baltimore, MD

Address Correspondence to:
Edward F. McCarthy, M.D.
Department of Pathology
Johns Hopkins Hospital
600 N. Wolfe Street
Baltimore, MD 21287
Phone: (410) 614-3655
Fax: (410) 614-3766
Email: mccarthy@jhmi.edu

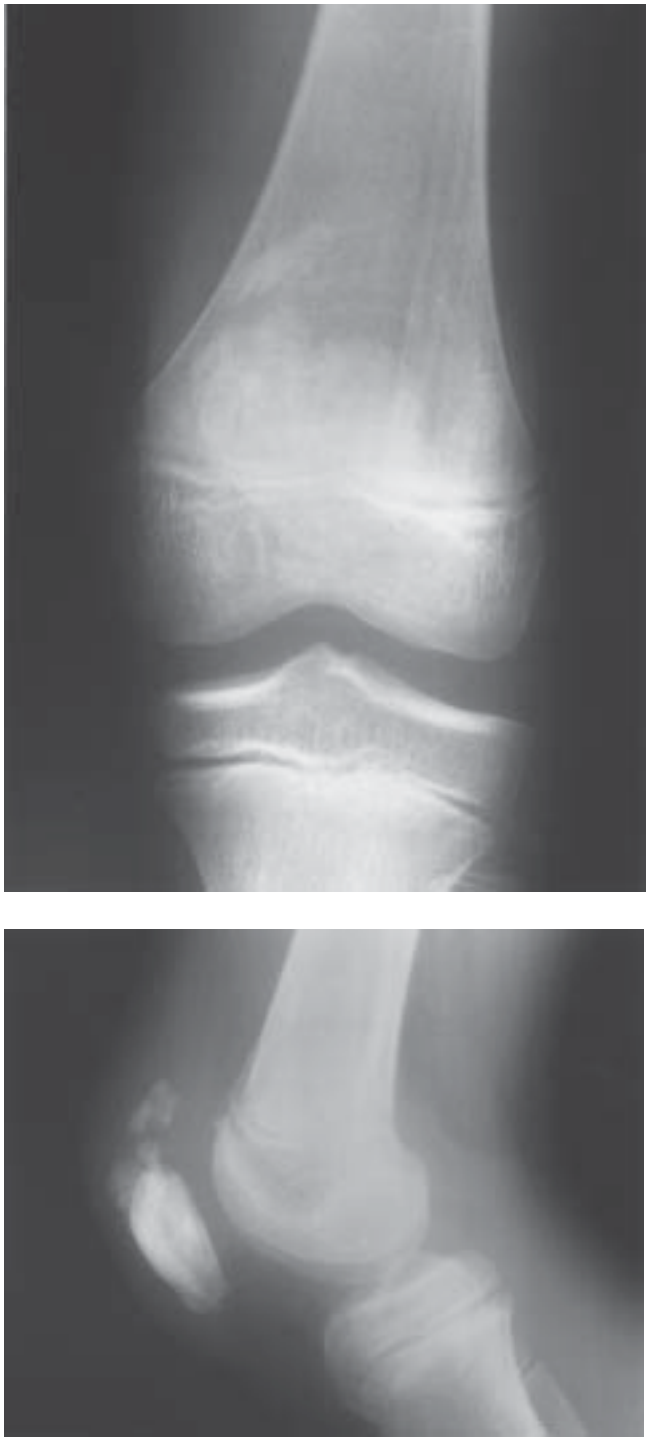

Figure 1. Case 1: Right knee of an 11 year old girl.
A) AP radiograph showing irregularity of the superior pole of the patella.
B) Lateral radiograph showing fragmentation of the superior pole with additional radiodensities due to cartilaginous repair.

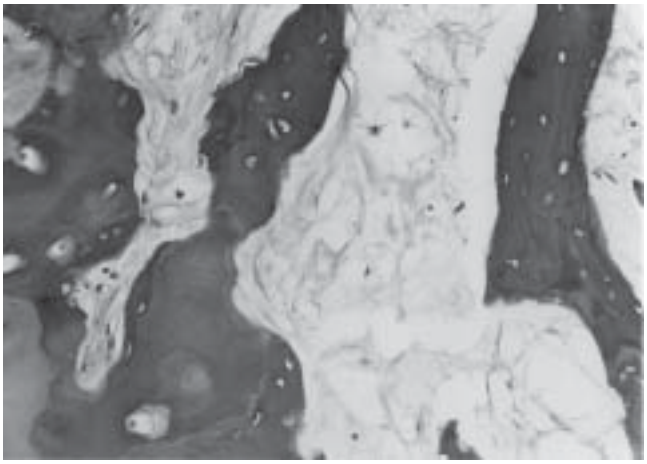

Figure 2. Photomicrograph (H & E x 160) of tissue removed from Case 1 showing necrotic bone and marrow.

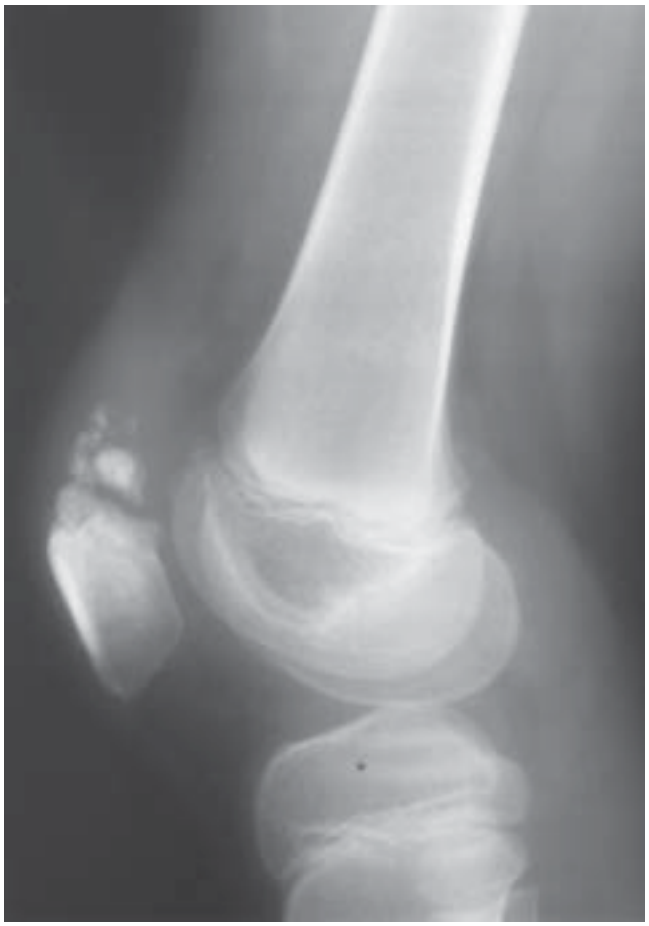

Figure 3. Lateral radiograph of right knee of 7 year old girl showing fragmentation of the upper pole the patella.

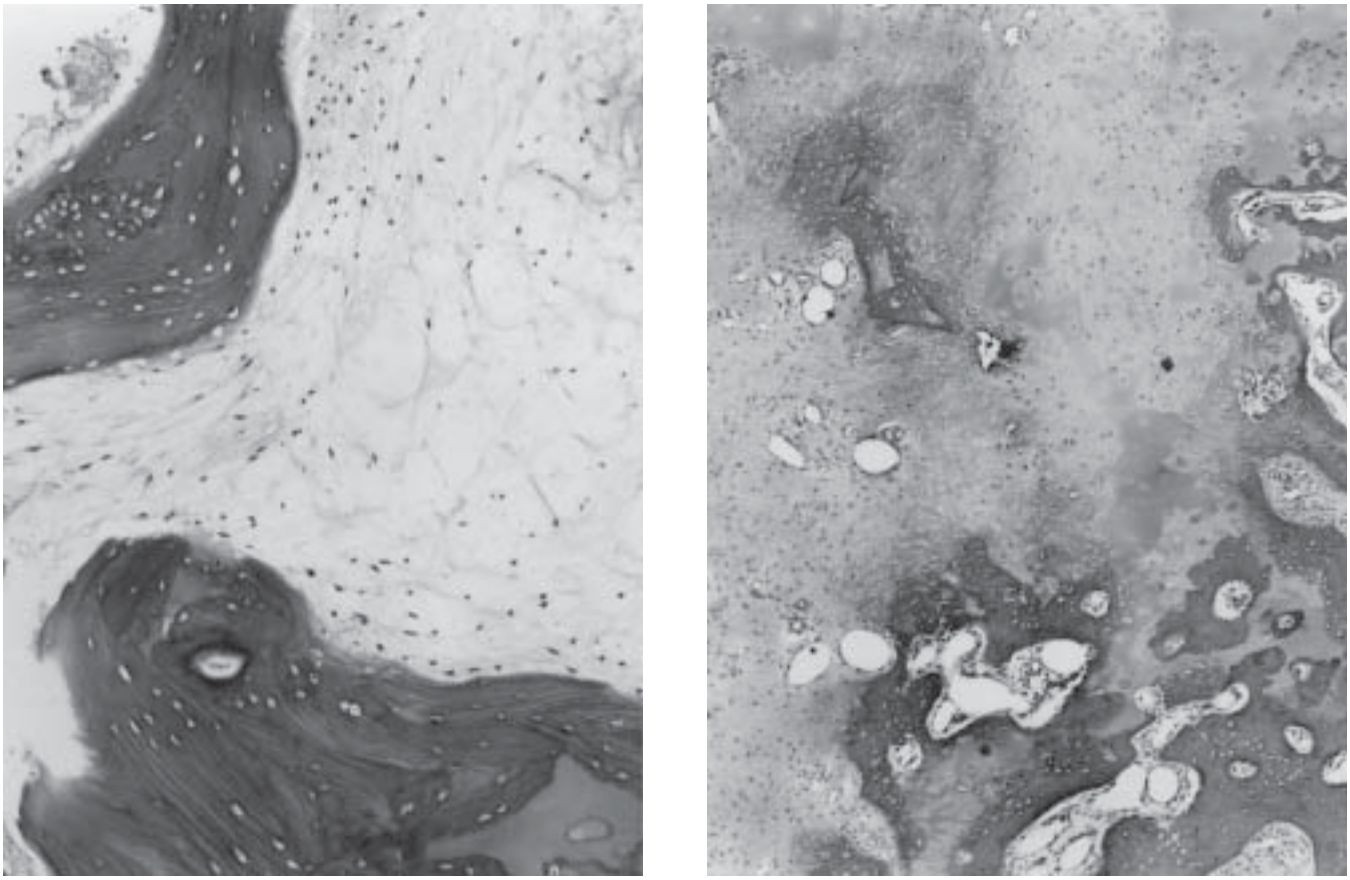

Figure 4. Histologic features of case 2.
A) Photomicrograph (H & E x 140) showing necrotic bone. The necrotic marrow shows early fibrosis.
B) Photomicrograph (H & E x 80) showing reparative bone cartilage and granulation tissue.

DISCUSSION

Fragmentation of the superior pole of the patella has been infrequently documented and the histologic features have not been described. Batten and Menelaus[1] reported six cases of superior pole fragmentation of the patella. They noted that none of the patients had symptoms localized to the proximal patella, but that all presented with clinical signs and symptoms consistent with either Osgood-Schlatter (OS) disease or Sinding-Larsen-Johansson (SLJ) disease. Four of the six patients had radiographic evidence of SLJ or OS disease either in the same knee or the opposite knee. Based on these findings, the authors suggested that similar processes take place in all three conditions and that the reported patients were predisposed to this process. All the patients were very active and ranged in age from ten to eleven years. None of the patients had any direct trauma to their knees.

In 1990, Grogen et al.[3] reported seven cases of patients with proximal pole fragmentation which they considered to be an avulsion injury as the result of direct trauma. All seven of their patients had a history of trauma to the knee. They interpreted these findings to be similar to other avulsion injuries to the patella, but they noted that injury to the proximal pole was the least common form of patellar avulsion. In addition to these two reports, several others have mentioned this syndrome in passing.[8, 12, 2]

In none of the reported cases have the histologic cases been documented. In our two cases the histologic features of the excised fragments showed changes similar to osteochondroses in other sites. In both cases there was extensive osteonecrosis and reparative change consisting of granulation tissue, new bone, and cartilage.

Multiple theories concerning the pathogenesis of traction osteochondroses have been proposed. Griswald and Hawley,[4] in their classic paper describing SLJ syndrome, suggested that repetitive trauma is a likely etiology. This theory, still the most widely accepted today, originates with studies of patients with spastic cerebral palsy who developed SLJ disease.[11,5] Kaye and Freiberger observed an extremely high incidence (28%) of SLJ syndrome in a population of cerebral palsy patients.[5] They postulated that SLJ disease in patients with CP was likely due to fatigue fractures. In 1977, Rosenthal and Levine[11] reported on seven more cases of SJL disease in 85 cerebral palsy cases—an incidence of only 5%. Four of these seven patients had evidence of OS disease as well. None had direct trauma to the knee. The authors concluded that SLJ disease in the setting of spastic CP was a result of excessive traction from the quadriceps in the setting of flexion contracture. In support of this theory, Perry and colleagues[10] found that for every degree of flexion, there was a 6% increase in quadriceps force needed to stabilize the knee. Therefore, in our patient (case 1) with spasticity and a five degree flexion contracture, there would be a 30% increase in quadriceps force applied to the knee. These forces could certainly be the stress needed to cause fragmentation of the patella.

Based on the study of child cadavers, it has been found that the patella likely begins ossifying in multiple foci beginning about age five or six.[7,9] As a result, ossification centers are separated by zones of cartilage. Skeletally immature individuals who subject growth centers to excessive strain from high levels of activity, muscle spasticity, or other unclear mechanisms can cause a traction osteochondrosis through these zones weakened by residual unossified cartilage. These injuries have previously been well characterized in the lower pole of the patella and in the tibial tuberosity. They are less well known in the superior pole of the patella. Therefore, osteochondrosis in this zone has not been given an eponym. Why osteochondrosis develops so rarely in the superior pole of the patella is unknown. The broader insertion of the quadriceps in this area probably distributes traction forces more evenly. Based on the histological findings of osteonecrosis in the two cases presented here, superior pole fragmentation is an osteochondrosis similar to the osteochondroses in most other locations.

REFERENCES

1. **Batten J, Menelaus MB**. Fragmentation of the proximal pole of the patella. Another manifestation of juvenile traction osteochondritis? *J Bone Joint Surg* [Br]. 1985;67(2):249-251.
2. **Crawford AH**. Fracture about the knee in children. *Orthop Clin North Am*. 1976;7:639-56.
3. **Grogen DP, Carey TP, Leffers D, Ogden JA**. Avulsion fractures of the patella. *J Pediatr Orthop* 1990;10(6):721-730.
4. **Hawley GW, Griswold AS**. Larsen-Johansson's disease of the patella. *Surg Gynecol Obstet*. 1928;47:68-72.
5. **Kay JJ, Freiberger RH**. Fragmentation of the lower pole of the patella in spastic lower extremities. *Radiology*. 1971;101:97-100.
6. **McCarthy EF, Frassica FJ**: Pathology of bone and joint disorders with clinical and radiographic correlation. Philadelphia: WB Saunders Company.1998;45-151.
7. **Ogden JA**. Radiology of postnatal skeletal development. Patella and tibial tuberosity. *Skeletal Radiol*. 1983;11:246-257.
8. **Ogden JA**. Skeletal injuries in the child. 2nd ed. Philadelphia: WB Saunders.1990;60-768.
9. **Ogden JA, McCarthy SM, Jokl P**. The painful bipartite patella. *J Pediatr Orthop*. 1982;2:263-269.
10. **Perry J, Daniel A, Ford W**. Analysis of knee-joint forces during flexed-knee stance. *J Bone Joint Surg*. 1975;57A:961-967.
11. **Rosenthal RK, Levin DB**. Fragmentation of the distal pole of the patella in spastic cerebral palsy. *J Bone Joint Surg* [Am]. 1977;59(7):934-939.
12. **Smillie IS**. Diseases of the knee joint, 2nd ed. Edinburgh: Churchill Livingstone. 1980;47.