Hip Pain in an 18-year-old Man

Arun Rajaram BA, Robert M. Tamurian MD,
John D. Reith MD, Charles H. Bush MD

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History and Physical Examination

An 18-year-old man presented with a 6-month history of left hip pain. There was no antecedent trauma but a gradual onset of pain that became persistent over the 6-month period. The patient's pain intensified with time and a bothersome limp on the left side was affecting his activity level. He had no other complaints or constitutional symptoms, nor did he report any chest pain or shortness of breath. His medical history was unremarkable. Family medical history was significant for B cell lymphoma in the patient's maternal grandfather.

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Each author certifies that his or her institution has approved the reporting of this case, that all investigations were conducted in conformity with ethical principles of research, and that informed consent for participation in the study was obtained.

A. Rajaram
The George Washington University School of Medicine,
Washington, DC, USA

R. M. Tamurian (✉)
Department of Orthopaedics and Rehabilitation, University of
Florida, PO Box 112727, Gainesville, FL 32611-2727, USA
e-mail: tamurrm@ortho.ufl.edu

J. D. Reith
Department of Pathology, University of Florida, Gainesville, FL,
USA

C. H. Bush
Department of Radiology, University of Florida, Gainesville, FL,
USA

On physical examination, the patient appeared healthy with stable vital signs and no apparent distress. No regional or distant lymphadenopathy and no soft tissue mass were present around the hip. He was tender to palpation in the left groin and greater trochanter. Range of motion of the hip revealed pain with rotation, greater with external than internal rotation. Flexion and extension did not produce pain. The neurovascular exam was normal, with 2 + dorsalis pedis and posterior tibial pulses. Motor examination was 5/5 and sensation was intact throughout all dermatomes.

Laboratory examination revealed an elevated white blood cell count of $12.7 \times 10^3/\mu\text{L}$, an elevated sedimentation rate of 21 mm/hour, and an elevated C-reactive protein of 15.4 mg/L.

Imaging studies included anteroposterior and lateral radiographs (Fig. 1), radionuclide Tc-99m methylene diphosphonate bone scan (Fig. 2), computed tomography (CT) scan (Fig. 3), and magnetic resonance imaging (MRI) (Fig. 4).

Based on the history, physical examination, laboratory studies, and imaging studies, what is the differential diagnosis?

Imaging Interpretation

Radiographs displayed a well-defined lesion with radio-dense margins within the epiphysis of the femoral head, with slight extension into the metaphysis. No substantial periosteal reaction was present (Fig. 1). The lesion showed marked uptake of radiotracer on bone scan (Fig. 2). CT scan showed a sharply marginated lesion in the proximal epiphyseal-metaphyseal region of the femur, and faint mineralization was visible within its matrix (Fig. 3).

Fig. 1 An anteroposterior radiograph of the left hip shows a well-defined lesion with radiodense margins in the epiphysis of the femoral head, with slight extension into the metaphysis.

Fig. 2 A delayed image from a Tc-99m methylene diphosphonate radionuclide bone scan shows marked uptake of radiotracer.

Coronal T1-weighted MRI scans through the hips displayed a lobular central lesion with a signal isointense or slightly hyperintense to surrounding muscle (Fig. 4A). The T2-weighted images revealed a lobular hyperintense lesion with low intensity septae and perilesional hyperintensity (flare phenomenon) and joint effusion (Fig. 4B).

Fig. 3 An axial CT image shows a well-demarcated lesion in the proximal epiphyseal-metaphyseal region of the femur, and faint intralesional mineralization is visible within the matrix.

Differential Diagnosis

- Aneurysmal bone cyst
- Chondroblastoma
- Chondromyxoid fibroma
- Clear cell chondrosarcoma
- Enchondroma
- Giant cell tumor
- Pigmented villonodular synovitis

An incisional biopsy of the lesion was performed and the histology of the lesion was examined (Fig. 5).

Based on the medical history, physical examination, laboratory studies, imaging studies, and histologic picture, what is the diagnosis and how should the patient be treated?

Histology Interpretation

Incisional biopsy revealed a cellular neoplasm composed of islands of immature cartilage surrounded by a proliferation of mononuclear cells and scattered multinucleated giant cells (Fig. 5A). The mononuclear cells had abundant cytoplasm with well-defined cytoplasmic borders, imparting a “cobblestone” appearance in areas (Fig. 5B). These cells possessed uniform nuclei, often containing a longitudinal groove. Calcification was prominent throughout the lesion and varied from coarse to “chicken wire” in appearance (Fig. 5C).

Fig. 4A–B (A) A coronal T1-weighted MR image shows a lobular lesion with a low intensity signal. (B) A coronal T2-weighted MR image reveals a lobular low intensity signal, with high intensity signal circumferentially in the perilesional bone marrow consistent with the flare phenomenon.

Diagnosis

Chondroblastoma of the proximal femur

Discussion and Treatment

Chondroblastoma is a rare, benign bone tumor originally described by Jaffe and Lichtenstein in 1942 [14]. Chondroblastomas account for 1% of primary bone tumors,

Fig. 5A–C (A) A medium-power view of the lesion reveals a cellular neoplasm composed of islands of immature cartilage surrounded by a proliferation of mononuclear cells and scattered multinucleated giant cells (Stain, hematoxylin and eosin; original magnification, $\times 200$). (B) A high-power view of the lesion displays mononuclear cells with abundant cytoplasm and well-defined cytoplasmic borders, imparting a cobblestone appearance (Stain, hematoxylin and eosin; original magnification, $\times 400$). (C) A high-power view of the lesion displays prominent calcification that varies from coarse to chicken wire in appearance (Stain, hematoxylin and eosin; original magnification, $\times 400$).

occur most often in the epiphyses of long bones, and occasionally extend into the metaphyses [12]. Lesions arise eccentrically and are rarely isolated to the metaphyseal or diaphyseal regions [1, 33]. Despite the predilection for secondary ossification centers, chondroblastomas may arise in small bones, such as the tarsals, or in the apophyses of bones, such as the iliac crest [6]. The lesions arise from immature chondroblasts and also contain foci of immature cartilage, osteoclastlike giant cells, and small areas of calcification [6]. The majority of chondroblastoma lesions are benign. However, cases of malignant transformation to a sarcoma (typically after radiation therapy), local recurrence, and lung metastases have been reported in the literature [15].

As with any reactive lesion of bone, infection should be considered as a possible cause of the patient's condition. Radiographically, the first finding of pyogenic osteomyelitis is a subtle radiolucency in the area of inflammation [9]. Approximately 2 weeks after the onset of infection, a small section of reactive bone around the inflammation becomes visible. The primary radiographic feature that distinguishes osteomyelitis from neoplasms is the irregular or serpiginous border often seen around infections [9]. Given the well-defined, geographic, regular border demarcating the lesion in our case, infection was not given strong consideration when correlated with the patient's presenting complaints and lack of systemic symptoms.

Aneurysmal bone cysts are benign lesions with cystlike walls, mostly consisting of cellular fibrous septa separated by blood. Long-bone metaphyseal location of the lesion can also aid in diagnosis of aneurysmal bone cyst, as most chondroblastomas are found in an epiphyseal or apophyseal location [18, 23]. Aneurysmal bone cysts make up over 2% of all bone tumors and 85% of all patients affected are younger than 20 years [23]. Up to 50% of these tumors are secondary to other reactive lesions, such as chondroblastomas [23]. In addition, underlying lesions may not be visible due to the rapid growth and distortion by the cysts. Because these cysts occur most often in pediatric and adolescent patients, they belong in the differential diagnosis in this age group. In young individuals, when an aneurysmal bone cyst in the epiphyses has enlarged and nearly replaced the primary lesion, meticulous histologic study and S100 staining should be used to identify a primary lesion [23]. In our patient, the histologic presence of chondroblasts and a positive S100 stain ruled out a primary cyst and confirmed the diagnosis of a chondroblastoma.

Chondromyxoid fibroma is a benign, cartilaginous neoplasm composed primarily of myxoid cartilage and accounts for less than 1% of all primary bone tumors. Lesions are most commonly located in the metaphyses of the major long bones, with a predilection for the proximal tibia [7]. Chondromyxoid fibroma mainly affects

individuals in the second or third decades of life and has a slight male predominance [7]. Chondromyxoid fibroma and chondroblastoma can be difficult to differentiate both clinically and pathologically since they affect relatively the same age group and share histologic features, such as numerous giant cells and chondroblastic differentiation. However, chondromyxoid fibroma is usually located in the metaphyses, lacks microscopic calcifications between the chondroblasts, and is characterized by stellate cells found clustered within the lesion [5, 7]. The lesion in our patient was located at the epiphyses of the femur, possessed microscopic "chicken wire" calcifications, and lacked the characteristic stellate cells found in chondromyxoid fibroma, thus, ruling out the diagnosis of chondromyxoid fibroma.

Clear cell chondrosarcoma is a rare, malignant tumor that represents around 0.2% of all primary bone tumors. These tumors commonly arise in the epiphysis and 59% involve the proximal femur [25]. Clear cell chondrosarcoma has been reported in patients as young as 14 years; however, it primarily affects individuals older than 20 years, with a mean age of 39 years [24, 25]. Clear cell chondrosarcoma and chondroblastoma can be difficult to differentiate radiographically because both are usually centered within the epiphysis and possess margins of sclerosis [24]. However, the characteristic features of clear cell chondrosarcoma that chondroblastoma lacks are enlarged clear cells and foci of reactive bone trabeculae [24, 25], neither of which were present in our patient.

Enchondroma is a benign, cartilaginous lesion that typically arises in the medullary canal of tubular bones and accounts for 3% to 10% of all primary bone tumors. Solitary enchondromas are found in the metacarpals or phalanges in approximately 50% of the cases, and 20% of the lesions involve the proximal or distal femur [32]. Enchondromas affect both males and females equally and usually occur in individuals between 20 and 40 years of age. Solitary enchondromas are most commonly asymptomatic and are usually an incidental finding when the area is radiographed for another purpose [8, 32]. They are not typically located in an epiphysis. Pathologically, these lesions consist of small clusters of proliferating, but differentiated chondrocytes surrounded by areas of hypocellular, basophilic matrix. Moreover, these clusters contain areas where two chondrocytes are present within a single lacuna, mimicking the appearance of a binucleated cell [8]. However, in our patient, the presence of symptoms, preponderance of immature chondroblasts, and the absence of the "binucleated" chondrocytes eliminated the diagnosis of enchondroma.

Giant cell tumors often occur in the epiphyseal-metaphyseal portion of long bones and are appropriate for the differential diagnosis. Demographically, giant cell tumors

affect females more frequently than males (1.3:1), and 98% of giant cell tumors occur in individuals older than 25 years with closed physes [26]. Most adolescent giant cell tumor cases occur in females due to earlier skeletal maturation. Radiographically, giant cell tumors differ from chondroblastomas as purely radiolucent lesions without the intralesional calcification present in chondroblastomas. Additionally, the flare phenomenon found in chondroblastomas is less prominent in giant cell tumors [13, 17, 24].

Histologically, the main differentiating characteristic is cartilage matrix. Giant cell tumors rarely contain this feature. The mononuclear cells in giant cell tumor and chondroblastoma are histologically similar, but the cobblestone appearance of the chondroblasts is absent in a giant cell tumor. Chondroblastomas usually occupy less than ½ of the epiphyses, whereas giant cell tumors often involve a much larger portion of the epiphyses [24]. Immunohistochemistry is a key differentiating feature, with giant cell tumors staining negative with S100 and chondroblastomas staining positive [18, 28, 31].

Pigmented villonodular synovitis is a benign process that originates in synovial tissues predominantly affecting the knee, hip, shoulder, and ankle. Symptoms include increasing joint pain with decreasing range of motion and mobility [10, 27]. Pigmented villonodular synovitis primarily affects individuals between the ages of 20 and 50 years, with a slight male predominance. One characteristic feature of pigmented villonodular synovitis is a tendency to affect both sides of the joint. In the hip, pigmented villonodular synovitis typically produces marked erosion of the femoral head, neck, and acetabulum [10, 27]. Erosions in the hip occur early in the course of the disease due to the constrained joint capsule that is unable to expand in response to the enlarging synovium [11]. MRI is very useful in diagnosing pigmented villonodular synovitis because of the lobulated synovial mass elevating the joint capsule and hemosiderin-induced low signal intensity on T1- and T2-weighted sequences. These imaging findings, present in over 95% of the cases [3], distinguish pigmented villonodular synovitis from chondroblastoma.

Individuals affected by chondroblastoma are usually adolescents. Many lesions have been discovered in open, closing, and closed epiphyseal growth plates [29]. Seventy-five percent of lesions occur in patients who are in the second decade of life, with a male to female incidence of 2.5:1 [21, 24, 33]. Over 70% of the lesions involve the long bones, with the proximal humerus being the most common location. Chondroblastoma typically arises as a solitary lesion; however, in rare cases, polyostotic lesions may affect two adjacent bones, such as the talus and calcaneus [6]. Because 98% of the lesions originate within the epiphyses, the radiographic location of the chondroblastoma is the critical diagnostic feature [24].

The most common presenting complaints are aching, intermittent pain and swelling of the adjacent joint. Decreased motion and pain gradually increase with time. Pathologic fracture, as the primary manifestation, is extremely rare [4, 6]. In ⅓ of the patients, chronic reactive synovitis may lead to joint effusion, especially in patients with lesions affecting the knee. Symptoms can persist for several months to years before patients seek treatment [6, 24]. Bloem and Mulder [2] reported 43% of 104 patients presented with pain as the sole complaint and 31% of the patients presented with pain and impaired joint function. Aside from the possible slight elevation in erythrocyte sedimentation rate, laboratory test results are usually unremarkable [24].

Radiographically, chondroblastomas appear as radiolucent, round lytic lesions, usually arising in a secondary ossification center. The tumor has a sharply defined sclerotic margin consisting of mature, reactive bone [2, 6]. Turcotte et al. [30] observed well-defined borders in 74% of 46 patients. Lobulation and centrally radiodense areas may be found in the lytic regions, reflecting the chondroid matrix calcification that is radiographically visible in 25% of the lesions [22, 30]. Most chondroblastomas are 1 to 4 cm in diameter, but quite extensive lesions larger than 10 cm have been reported [6, 20, 33].

Chondroblastomas display increased uptake on bone scan corresponding to the radiographic extent of the lesion. Increased uptake is expected due to the matrix-mineralizing metabolic activity of the lesion. Accurate evaluation of the bone scan can be obscured due to the increased uptake by adjacent physal activity [6, 33]. Angiography is rarely indicated but will display a hypervascular lesion confined within the radiographic margin [6].

CT can be useful in estimating the relationship of the lesion to the articular surface and the physal plate. Additionally, CT scans will exhibit the punctate calcifications of the lesions [6, 33].

MR images are useful in judging the size of the chondroblastoma, but the signal intensity of the lesion varies between cases [6, 16]. Chondroblastomas usually demonstrate a low-intensity signal on T1-weighted images. As with any cartilaginous lesion, chondroblastomas can demonstrate mixed signal intensity on T2-weighted images. Areas consisting of mature cartilage will demonstrate high signal intensity due to the high water content. In addition, low intensity signal areas may be seen due to calcification, immature chondroid matrix, and hemosiderin [16, 19]. T2-weighted images are also useful in showing the frequent presence of perilesional marrow edema, which displays the characteristic flare phenomenon often seen with chondroblastomas. In those cases that have metaphyseal extension, it is not uncommon to see some degree of periosteal reaction given the reactive nature of chondroblastoma [17].

Upon gross examination, the lesion appears as a soft, meaty, vascular tissue that lacks evidence of its cartilaginous origin [6]. Chondroblastomas appear pinkish-gray, as opposed to the bluish-gray appearance of most cartilaginous tumors. Evidence of nearly complete trabecular bone destruction can be seen in the areas occupied by the tumor [24, 33]. Additionally, the peripheral borders of adjacent bone may display increased density because of the deposition of a sclerotic outer layer of reactive bone. In about 20% of the tumors, tissue may be seen extending into the adjacent joint tissue, usually through the cruciate ligaments of the knee or the ligamentum teres of the hip [29]. When an intact, completely excised specimen is studied, a central chondroid lesion is seen, which often contains a well-demarcated cyst [13]. Also, 10% to 15% of lesions can be adjacent to blood-filled cavities, which represent secondary aneurysmal bone cysts [6, 18, 24].

Histologically, the background stroma contains large, polygonal cells, with basophilic nuclei centrally located within an eosinophilic cytoplasm. One of the characteristic features of chondroblastomas is that the cells mold together in a manner that resembles a mosaic tile or cobblestone pattern. These cells have been identified as chondroblasts [6, 18]. Dispersed throughout the tissue are islands of immature cartilage and multinucleated giant cells. The cartilaginous islands possess chondrocytes with light, basophilic-staining matrices. Calcifications can occur within the cartilage islands or within the chondroblasts and can appear coarse or can envelope individual cells in the characteristic “chicken wire” pattern [6, 13, 18]. These clumps account for the small specks of density seen on radiographs in around 50% of the tumors [24]. Foci of spindle cells also may occur in the lesion due to

fibrohistiocytic changes after hemorrhage. Little mitotic activity is seen in chondroblastomas [18, 24, 33].

In regard to natural history, most lesions behave as active, Enneking Stage 2, symptomatic processes with slow growth. Extension from the epiphysis to the metaphysis across the physeal plate is not uncommon [6]. If left untreated, a minority of chondroblastomas may progress to Stage 3, with extension into adjacent soft tissue or the joint cavity. Malignant transformation to chondrosarcoma is rare but has been associated with lesions in the pelvic girdle and low doses of radiation therapy [6].

Treatment of chondroblastomas is required for attempted cure since no evidence has shown that these lesions resolve spontaneously. Chemotherapy is not indicated, and radiation therapy is contraindicated because of the resulting risk of malignant transformation [6]. Surgical treatment via curettage and bone grafting is the most common procedure used to treat chondroblastomas. The recurrence rate for Stage 2 lesions is approximately 10% [6].

An open biopsy was performed on our patient through a limited incision, with care being taken to avoid tissue contamination and tumor spillage. After frozen-section pathologic examination excluded malignancy and the diagnosis of chondroblastoma was confirmed, the lesion was treated with thorough curettage, bone grafting with cancellous allograft, and prophylactic internal fixation of the femoral head and neck. Postoperatively, the patient did well with protected weightbearing and progressed to full weightbearing 4 weeks after surgery. At 3 months postoperatively, he had full range of motion and a pain-free hip and was walking without assistive devices. Plain radiographs (Fig. 6) of his femur demonstrated early incorporation of the bone graft without evidence of

Fig. 6A–B (A) Anteroposterior and (B) lateral radiographs of the left hip show stable internal fixation with incorporation of bone graft.

recurrent tumor. At last contact, 20 months after surgery, our patient has returned to swimming and his prior activity level without difficulty. He has full range of motion and remains pain-free. There is no evidence of recurrence of the tumor, and his hardware is in an unchanged position.

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References

- Azorín A, González-Mediero I, Colmenero I, De Prada I, López-Barea F. Diaphyseal chondroblastoma in a long bone: first report. *Skeletal Radiol*. 2005;35:49–52.
- Bloem JL, Mulder JD. Chondroblastoma: a clinical and radiological study of 104 cases. *Skeletal Radiol*. 1985;14:1–9.
- Bravo SM, Winalski CS, Weissman BN. Pigmented villonodular synovitis. *Radiol Clin North Am*. 1996;34:311–326.
- Cohen J, Cahen I. Benign chondroblastoma of the patella: a case report. *J Bone Joint Surg Am*. 1963;45:824–826.
- Dahlin DC. Chondromyxoid fibroma of bone, with emphasis on its morphological relationship to benign chondroblastoma. *Cancer*. 1956;9:195–203.
- Enneking WF. Chondroblastoma. *Clinical Musculoskeletal Pathology*. Gainesville, FL: University of Florida; 1998:12:17–20.
- Enneking WF. Chondromyxoid fibroma. *Clinical Musculoskeletal Pathology*. Gainesville, FL: University of Florida; 1998:12:21–24.
- Enneking WF. Enchondroma. *Clinical Musculoskeletal Pathology*. Gainesville, FL: University of Florida; 1998:12:11–15.
- Enneking WF. Osteomyelitis, pyogenic. *Clinical Musculoskeletal Pathology*. Gainesville, FL: University of Florida; 1998:5:1–6.
- Enneking WF. Pigmented villonodular synovitis. *Clinical Musculoskeletal Pathology*. Gainesville, FL: University of Florida; 1998:9:9–13.
- Frassica FJ, Bhimani MA, McCarthy EF, Wenz J. Pigmented villonodular synovitis of the hip and knee. *Am Fam Physician*. 1999;60:1404–1410.
- Hameed MR, Blacksin M, Das K, Patterson F, Benevenia J, Aisner S. Cortical chondroblastoma: report of a case and literature review of this lesion reported in unusual locations. *Skeletal Radiol*. 2005;35:295–297.
- Huvos AG. Chondroblastoma. *Bone Tumors: Diagnosis, Treatment, and Prognosis*. Philadelphia, PA: WB Saunders; 1991:295–314.
- Jaffe HL, Lichtenstein L. Benign chondroblastoma of bone: a reinterpretation of the so-called calcifying or chondroblastomatous giant cell tumor. *Am J Pathol*. 1942;18:969–992.
- Jambhekar NA, Desai PB, Chitale DA, Patil P, Arya S. Benign metastasizing chondroblastoma: a case report. *Cancer*. 1998;82:675–678.
- Jee WH, Park YK, McCauley TR, Choi KH, Ryu KN, Suh JS, Suh KJ, Cho JH, Lee JH, Park JM, Lee YS, Ok IY, Kim JM. Chondroblastoma: MR characteristics with pathologic correlation. *J Comput Assist Tomogr*. 1999;23:721–726.
- Kaim AH, Hügli R, Bonél HM, Jundt G. Chondroblastoma and clear cell chondrosarcoma: radiological and MRI characteristics with histopathological correlation. *Skeletal Radiol*. 2002;31:88–95.
- Kang LC, Chamyan G, Barness-Gilbert I. Pathology teach and tell: chondroblastoma. *Pediatr Pathol Mol Med*. 2002;21:71–74.
- Keenan S, Bui-Mansfield LT. Musculoskeletal lesions with fluid-fluid level: a pictorial essay. *J Comput Assist Tomogr*. 2006;30:517–24.
- Kricun ME, Kricun R, Haskin ME. Chondroblastoma of the calcaneus: radiographic features with emphasis on location. *Am J Roentgenol*. 1977;128:613–616.
- Lin PP, Thenappan A, Deavers MT, Lewis VO, Yasko AW. Treatment and prognosis of chondroblastoma. *Clin Orthop Relat Res*. 2005;438:103–109.
- McLeod RA, Beabout JW. The roentgenographic features of chondroblastoma. *Am J Roentgenol Radium Ther Nucl Med*. 1973;118:464–471.
- Mirra J. Aneurysmal bone cyst. *Bone Tumors: Clinical, Radiologic, and Pathologic Correlations*. Philadelphia, PA: Lea and Febiger; 1989:1267–1309.
- Mirra J. Chondroblastoma. *Bone Tumors: Clinical, Radiologic, and Pathologic Correlations*. Philadelphia, PA: Lea and Febiger; 1989:589–623.
- Mirra J. Clear cell chondrosarcoma. *Bone Tumors: Clinical, Radiologic, and Pathologic Correlations*. Philadelphia, PA: Lea and Febiger; 1989:535–546.
- Mirra J. Giant cell tumors. *Bone Tumors: Clinical, Radiologic, and Pathologic correlations*. Philadelphia, PA: Lea and Febiger; 1989:941–623.
- Mirra J. Pigmented villonodular synovitis invading bone. *Bone Tumors: Clinical, Radiologic, and Pathologic Correlations*. Philadelphia, PA: Lea and Febiger; 1989:1766–1775.
- Sotelo-Avila C, Sundaram M, Kyriakos M, Gravis E, Taylor A. Case report 373: diaphyseal chondroblastoma of the upper portion of the left femur. *Skeletal Radiol*. 1986;15:387–390.
- Springfield DS, Capanna R, Gherlinzoni F, Picci P, Campanacci M. Chondroblastoma: a review of seventy cases. *J Bone Joint Surg Am*. 1985;67:748–755.
- Turcotte RE, Kurt AM, Sim FH, Unni KK, McLeod RA. Chondroblastoma. *Hum Pathol*. 1993;24:944–949.
- Unni KK. Chondrogenic tumors. *Dahlin's Bone Tumors: General Aspects and Data on 11,087 Cases*. Philadelphia, PA: Lippincott-Raven; 1996:4–9.
- Unni KK. Enchondroma. *Dahlin's Bone Tumors: General Aspects and Data on 11,087 Cases*. Philadelphia, PA: Lippincott-Raven; 1996:25–35.
- Vigorita VJ. Chondroblastoma. *Orthopaedic Pathology*. Philadelphia, PA: Lippincott Williams and Wilkins; 1999:363–367.