Osteoid Osteoma and Osteoblastoma

Abstract

Osteoid osteoma and osteoblastoma are commonly seen benign osteogenic bone neoplasms. Both tumors are typically seen in the second decade of life, with a notable predilection in males. Histologically, these tumors resemble each other, with characteristically increased osteoid tissue formation surrounded by vascular fibrous stroma and perilesional sclerosis. However, osteoblastomas are larger than osteoid osteomas, and they exhibit greater osteoid production and vascularity. Clinically, osteoid osteoma most commonly occurs in the long bones (e.g., femur, tibia). The lesions cause night pain that is relieved with nonsteroidal anti-inflammatory drugs (NSAIDs). Osteoblastoma is most frequently located in the axial skeleton, and the pain is usually not worse at night and is less likely to be relieved with NSAIDs. Osteoblastoma can be locally aggressive; osteoid osteoma lacks growth potential. Osteoid osteoma may be managed nonsurgically with NSAIDs. When surgery is required, minimally invasive methods (e.g., CT-guided excision, radiofrequency ablation) are preferred. Osteoblastoma has a higher rate of recurrence than does osteoid osteoma, and patients must be treated surgically with intralesional curettage or en bloc resection.

Osteoid Osteoma

Osteoid osteoma was first described in 1935 by Jaffe, who also coined the term. It is the most commonly seen benign bone-forming lesion, accounting for 10% to 12% of all benign bone tumors and 3% of all primary bone tumors. This lesion most commonly occurs in persons aged 5 to 25 years, with a male:female ratio of 2:1. In >50% of cases, the lesion occurs in the metaphysis and diaphysis of the long bones, particularly the femur and tibia. Other anatomic sites of involvement include the spine, upper extremity, pelvis, sacrum, ribs, hands, and feet (Table 1).

Gross Structure and Histology

Osteoid osteoma is a solitary lesion, typically <1.5 cm in diameter and contains a discrete central area known as the nidus that is surrounded by dense sclerotic bone tissue. In general, osteoid osteoma is a solitary le-
sion; in rare cases, however, more than one nidus may be circumscribed by a single block of sclerotic bone.9,10

Microscopically, the nidus is composed of thin seams of osteoid or woven bone lined with osteoblasts, which represents a process of bone remodelling with osteoblastic activity (Figure 1). Osteoclastic bone resorption occurs simultaneously and gives rise to a clearer area at the periphery of the circular nidus. The nidus is surrounded by a region of active bone formation that appears as sclerotic dense bone with various patterns of maturation.1,4

Osteoid osteoma does not grow or behave in a locally aggressive manner, and it has no potential for malignant transformation.4,6

### Biology and Pathophysiology

Schulman and Dorfman11 demonstrated abundant nerve fibers within the nidus matrix adjacent to areas rich in arterioles. High levels of prostaglandin synthesis in the nidus of osteoid osteoma have been reported in several other studies.12,13 This finding was supported by studies demon-

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**Table 1**

<table>
<thead>
<tr>
<th>Features of Osteoid Osteoma and Osteoblastoma</th>
<th>Osteoid Osteoma</th>
<th>Osteoblastoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence</td>
<td>≈12% of all benign bone tumors</td>
<td>≈3% of all benign bone tumors</td>
</tr>
<tr>
<td>Age</td>
<td>5 to 25 yr</td>
<td>10 to 25 yr</td>
</tr>
<tr>
<td>Sex</td>
<td>Male:female ratio of 2:1</td>
<td>Male:female ratio of 2:1</td>
</tr>
<tr>
<td>Size</td>
<td>&lt;2 cm in diameter (typically &lt;1.5 cm)</td>
<td>&gt;2 cm in diameter (average, 3.5 to 4 cm)</td>
</tr>
<tr>
<td>Location</td>
<td>&gt;50% of lesions in the lower extremity long bones (ie, femur, tibia). Other common sites: spine, upper extremity, hands, feet, and pelvis</td>
<td>&gt;35% of lesions in the vertebral column (posterior elements). Other common sites: long bones, craniofacial bones, hands, and feet</td>
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<tr>
<td>Clinical features</td>
<td>Local pain that is most severe at night and can be relieved with nonsteroidal anti-inflammatory drugs. Depending on location, patients may present with bone deformity, gait disturbance, limb-length discrepancy, or synovitis.</td>
<td>Dull, aching, progressive local pain. Due to higher incidence of spinal involvement, patients may experience neurologic symptoms, scoliosis, or torticollis. Local tenderness and swelling may be seen.</td>
</tr>
<tr>
<td>Imaging findings</td>
<td>Plain radiograph is obtained initially, but supportive imaging is required. Bone scan: Sensitive and valuable in localizing the lesion. Shows high uptake. CT: Imaging method of choice. Shows the low-attenuated nidus with surrounding sclerosis. MRI is controversial: Nonspecific findings with frequent misinterpretation.</td>
<td>Plain radiograph obtained initially. Lesions are larger. Supportive imaging is required. Bone scan: Sensitive. Shows high uptake of radionuclide at the lesion site. CT: Imaging method of choice. Larger lesion, central mineralization, expansile bone growth, less reactive sclerosis, thin marginal bone shell. MRI is controversial: Nonspecific findings with overestimation of tumor extent and nature.</td>
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<tr>
<td>Histology and nature</td>
<td>Central nidus composed of tiny osteoid islands lined by osteoblasts. The area peripheral to the nidus appears clearer because of osteoclastic resorption. Dense sclerotic bone surrounds the nidus. Benign. No growth potential.</td>
<td>Centrally, lesions demonstrate a less organized osteoid pattern than osteoid osteoma, with greater vascularity. Bony trabeculae lined by osteoblasts. Presence of epithelioid osteoblasts indicates aggressiveness. Less sclerotic bone with a thin shell of newly formed periosteal bone at the margin. Benign. Growth is localized, with aggressive potential.</td>
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Demonstrating the expression of cyclooxygenase-1 and cyclooxygenase-2 isozymes by the tumor tissue; both enzymes are responsible for protein processing in the prostaglandin biosynthesis pathway. These reports suggested an important pathophysiologic role for prostaglandins both as mediators of pain and vasodilation that may stimulate the nerve endings by increasing the blood flow within the tumor.

**Clinical Features**

The most prominent clinical symptom of osteoid osteoma is the presence of local pain that is typically more frequent and severe at night and that is relieved with administration of nonsteroidal anti-inflammatory drugs (NSAIDs). Other signs and symptoms are local swelling and tenderness, bony deformity, gait disturbances, and muscle atrophy; depending on the proximity to a joint, the clinician may note effusion, synovitis, degenerative changes, limitation of movement, and contractures. Particularly in pediatric patients, osteoid osteoma may present with rapidly progressive painful scoliosis when the spinal column is involved. In most cases, the concavity of the scoliotic curve is ipsilateral to the lesion as a result of muscle spasm and pain. Limb-length discrepancy may be associated with pediatric osteoid osteoma. Peyser et al reported average limb-length discrepancy of 12 mm in four pediatric patients with osteoid osteoma of the femur and tibia. In each case, the involved extremity was longer than the uninvolved extremity. One possible explanation for limb overgrowth in children with osteoid osteoma may be the resulting inflammatory response and associated hyperemia, especially in patients with lesions located near the open growth plate.

**Diagnostic Imaging**

The typical pain pattern and physical findings related to the location of the tumor often leads the clinician to strongly consider a diagnosis of osteoid osteoma. A combination of plain radiographs, bone scintigraphy, CT, and occasionally MRI, is usually sufficient to confirm the diagnosis.

**Radiography**

Conventional radiography is the initial examination of choice. A plain radiograph of osteoid osteoma may reveal a characteristic oval radiolucency representing the nidus as well as a surrounding area of reactive bone sclerosis with or without periosteal bone formation (Figure 2, A). However, these findings are typical of cortical lesions, and they may not be found on a plain radiograph if the lesion is located either in the intramedullary cavity or in areas of the skeleton that are difficult to assess with radiography alone (eg, spine, pelvis, small bones of the hands and feet).

**Scintigraphy**

Bone scintigraphy is a highly sensitive diagnostic modality for detecting and localizing osteoid osteoma. These images demonstrate the classic markedly increased radionuclide uptake by the lesion in the affected foot, at the left, and no abnormal findings in the contralateral foot at the right. (Panel A adapted with permission from Peyser A, Appelbaum Y: Radiofrequency ablation of bone tumors. *Current Orthopaedic Practice* 2009;20[6]:616-621.)
uptake by the nidus (Figure 2, B). Nuclear medicine bone scans use technetium Tc-99m-labeled diphosphonates, which have avidity for areas with increased osteoblastic activity and bone turnover. In addition, portable gamma cameras can be used as radiotracer detectors intraoperatively to localize the nidus during resection. Despite its increased sensitivity, scintigraphy is not a specific imaging method for determining the distinctive features of the lesions.

CT
CT is considered to be the imaging method of choice for visualizing the anatomic position of the nidus and aiding in the differential diagnosis. The characteristic appearance of osteoid osteoma on thin-slice CT scan is of a low-attenuation nidus with central mineralization and varying degrees of sclerosis surrounding the nidus (Figure 3). CT imaging is particularly useful when the nidus is in an intra-articular location or is not apparent radiographically because of the presence of complex anatomic features.

MRI
The effectiveness of MRI in diagnosing osteoid osteoma is controversial. The appearance of the lesion may be highly variable, and the presence of associated soft-tissue changes and bone marrow edema may result in diagnostic errors. Davies et al retrospectively reviewed the MRI findings of 43 patients with osteoid osteoma and compared the accuracy of MRI localization with that of other imaging modalities. Six tumors were not seen on MRI, and nine were poorly visualized. The potential for a missed diagnosis was 35% based solely on the MRI findings.

Management and Prognosis

Nonsurgical
Nonsurgical management with salicylates or NSAIDs is a justifiable therapeutic option because these drugs can effectively relieve pain, which is typically the presenting and most striking patient complaint. However, few published studies have evaluated the results of prolonged medical management and reported complete resolution of symptoms with discontinuation of NSAIDs. Kneisl and Simon noted an average time of 33 months to resolution of symptoms in six patients treated with NSAIDs. Sporadic case reports in the literature describe the probability of the evolution of osteoid osteoma into osteoblastoma after prolonged nonsurgical management with NSAIDs.

Surgical
Surgical management is warranted in cases in which the pain is severe and...
unresponsive to medication. Surgery is also warranted for patients who are unwilling to endure pain and accept long-term medical treatment because of potential gastrointestinal complications associated with the use of NSAIDs. Prolonged presence of osteoid osteoma lesions, especially in skeletally immature patients, may lead to complications such as growth disturbances, scoliosis, and osteoarthritis.\textsuperscript{27} The most commonly used surgical management techniques include open excision of the lesion, CT-guided percutaneous excision, and CT-guided radiofrequency (RF) ablation.

**Open Excision**

Until the late 1990s, open excision was the only surgical option.\textsuperscript{16} En bloc excision of the tumor, and cortical shaving and curettage of the nidus cavity, are frequently used conventional techniques with successful outcomes.\textsuperscript{24,27} However, these techniques can be unexpectedly challenging for both surgeon and patient. The tumor may be difficult to identify intraoperatively, and incomplete removal may result in recurrence. Additionally, resection of weight-bearing bone may necessitate prolonged hospital stay as well as restrictions on activities and weight bearing.\textsuperscript{15,28,29} Advances in imaging

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Figure 4: Treatment algorithm for osteoid osteoma and osteoblastoma.

- **Osteoid Osteoma**: Nonsurgical management with NSAIDs
- **Osteoblastoma**: Surgical management

**Unrelieving or Worsening Pain**: Complications (eg, scoliosis)
- **Surgical Management**
  - **Locations**: Vertebral column, close to peripheral nerves, fingers, toes, and carpal bones
- **Open Surgical Excision**
- **Minimally Invasive Procedures**
  - **CT-guided RF ablation**
  - **Other Techniques**

**Patient Preference is Surgery**
- **Contraindications to NSAIDs**
- **Nonaggressive Osteoblastomas in Expendable Bones**
- **Agressive and Large Lesions in Expendable Bones**

**Intralesional Curettage**
- ± cryotherapy or phenol + cementation or bone grafting

**En Bloc Resection**
- ± internal fixation and bone grafting (location dependent)

NSAIDs = nonsteroidal anti-inflammatory drugs, RF = radiofrequency
technology have made it possible to address the disadvantages of open excision by providing real-time CT guidance and percutaneous management of osteoid osteoma.

**CT-guided Percutaneous Excision**

In this technique, a cannulated curet is inserted into the lesion over a Kirschner wire under CT image guidance to excise the nidus. Fenichel et al.30 used this technique in a series of 18 patients with osteoid osteoma of the pelvis, femur, and tibia. Sixteen patients experienced immediate and permanent pain relief after the first procedure. In two patients, the nidus was missed because of misinterpretation of the CT scan, and no clinical improvement was observed postoperatively. In these patients, the procedure was repeated successfully, resulting in prompt postexcisional cessation of typical osteoid osteoma pain. There were two complications (femoral neurapraxia, skin abrasion), both of which resolved. Other authors have reported similar results with a high clinical success rate and minimal complications.31

**CT-guided Radiofrequency Ablation**

In CT-guided RF ablation, heat is applied locally to destroy the nidus (Figure 5). The RF probe is introduced into the nidus through a cannulated needle under CT-guided imaging, and the temperature at the tip of the probe is increased to approximately 90°C and maintained at that level for 5 to 6 minutes.32 The tip of the probe must be insulated to prevent injury to the soft tissues adjacent to the osteoid osteoma.

Rosenthal et al.29 performed CT-guided RF ablation on 263 patients with a mean age of 19 years. In total, 271 ablation procedures were performed: 249 for initial tumor treatment, 14 for recurrence after conventional surgery, and 8 for recurrence after prior RF ablation. All the procedures were technically successful, and two minor procedure-related complications were observed. After the procedure, all daily activities were resumed immediately. The only restriction involved avoidance of strenuous sports for 3 months for patients with lesions in weight-bearing bones. Of the 126 patients for whom 24-month follow-up data were available, 112 were pain-free and did not require additional procedures. The procedure was unsuccessful in nine patients, and the outcome was indeterminate in five. The authors suggested RF ablation as “the treatment of choice with high clinical success rate (91%), brief recovery, and gratifyingly low complication rate.”

Peyser et al.7 reported the results of CT-guided RF ablation in 22 pediatric patients with osteoid osteoma (mean age, 13 years 6 months). The procedure was technically successful in all patients, and all were discharged from the hospital within 24 hours postoperatively with no restrictions in weight bearing. At an average follow-up of 38.5 months, clinical recovery was achieved in all but two patients. These two patients underwent a second ablation, after which they remained symptom-free. The only procedure-related complication was observed in a patient with a nidus located in the tibial diaphysis. This patient experienced superficial skin infection of the ablation area that resolved with outpatient antibiotic treatment. The authors stated, “RF ablation in pediatric osteoid osteoma patients is a safe and effective technique.” Several other recent case series and cohort studies have demonstrated the effectiveness of RF ablation as a safe and minimally invasive method for the management of osteoid osteoma.33,34

In a series of 125 patients, Rosenthal et al.15 retrospectively compared the outcomes of RF ablation (38 patients) and surgical excision (87 patients). They found no significant difference between the two groups with regard to rate of recurrence ($P = 0.725$) and clinical success ($P = 0.722$). The average length of hospital stay was 0.18 day for the RF group and 4.7 days for the open excision group. No complications were associated with the percutaneous method. Two patients in the open excision group experienced complications, requiring a total of five secondary procedures.

No studies to date have compared CT-guided percutaneous excision with CT-guided RF ablation; however, clinical outcomes are known to be similar with both techniques. Higher success rates in tissue diagnosis have been reported with CT-guided excision than with RF ablation (69% to 77% and 47% to 50%, respectively), possibly because the entire nidus can be removed in the excision procedure.2,30,31 However, CT-guided excision of the nidus along with cortices may act as a stress riser and may predispose the patient to pathologic fracture or
activity-related restrictions postoperatively.

The current trend toward minimally invasive therapies has resulted in the use of many other less commonly employed percutaneous techniques for the management of osteoid osteoma. These include image-guided cryotherapy, drill trepanation with or without ethanol injection, thermal destruction by means of laser photocoagulation, and arthroscopic excision of juxta-articular lesions.\(^2,31,35\)

**Figure 6**

Schematic illustration demonstrating the spectrum of changes in lesion size and amount of sclerotic bone surrounding the lesion in osteoid osteoma and osteoblastoma. (Adapted with permission from Dorfman HD, Czerniak B: Benign osteoblastic tumors, in Dorfman HD, Czerniak B, eds: Bone Tumors. St. Louis, MO, Mosby, 1998, pp 85-127.)

Osteoblastoma was first described in 1932 by Jaffe and Mayer\(^36\) as “an osteoblastic-osteoid tissue forming tumor.” In 1956, Jaffe and Lichtenstein further characterized the lesion and independently proposed the term “benign osteoblastoma.”\(^37\) Osteoblastoma is a rare tumor that accounts for 3% of all benign bone tumors and approximately 1% of all primary bone tumors.\(^1,38\) Osteoblastoma most commonly arises in patients aged between 10 and 25 years, with a male:female ratio of 2:1; this is similar to the age and sex characteristics of patients with osteoid osteoma.\(^27,38\) Unlike osteoid osteoma, however, osteoblastoma is most often located in the posterior elements of the vertebral column and the sacrum. This presentation accounts for approximately one third of cases. Other commonly involved sites are the long bones (eg, femur, tibia), particularly within the medullary cavity and diaphysis. Osteoblastoma is also seen in the craniofacial bones (15%) and the hands and feet (14%) and, to a lesser extent, in sites such as the ribs, clavicle, and sternum.\(^2,7,38\)

**Gross Structure and Histology**

Grossly, osteoblastoma is considerably larger than osteoid osteoma, with an average diameter of approximately 4 cm.\(^5,37\) The tumor contains a larger and structurally less organized central area and a less dense sclerotic reaction circumscribing the lesion (Figure 6). The presence of more than one central zone per lesion (ie, multifocal osteoblastoma) is seen in 4% to 14% of cases, which is higher than in osteoid osteoma.\(^27,37,39\)

The microscopic features of osteoblastoma are similar to those of osteoid osteoma. Centrally, the lesion consists of an interlacing network of bony trabeculae within a loose fibrovascular stroma that is rimmed by a single row of benign osteoblasts that are responsible for osteoid formation (Figure 7). The lesions show a variable number of osteoclasts at the surfaces of the bony trabeculae; these osteoclasts are involved in bone resorption. The stroma of osteoblastoma demonstrates a less organized pattern of osteoid and trabecular bone distribution than does that of osteoid osteoma as well as greater vascularity. The lesion may show secondary aneurysmal bone cyst degeneration as a result of increased vascularity. The tumor is surrounded by a thin shell of newly formed periosteal bone tissue, which appears less sclerotic than that of osteoid osteoma and matures toward its periphery.\(^1,4,27\)

Osteoblastoma is a benign but locally aggressive tumor, with a clinical course ranging from slow, indolent progression to rapid aggressive growth. Aggressive osteoblastomas are associated with large epithelioid osteoblasts that rim the bony trabeculae, and they are more mitotically active than the cells in osteoid osteoma and conventional benign osteoblastoma.\(^1,4,6,27,38\) Zon Filippi et al\(^39\) demonstrated that a predominance of epithelioid osteoblasts is commonly seen in multifocal osteoblastomas (Figure 8).

Osteoblastoma has neither malignant nor metastatic potential. Histologically, the lesion has benign features even when it appears aggressive radiographically.\(^40\) Osteoblastoma that is reported to have malignant potential should be meticulously differentiated from other malignant bone tumors, in particular, from the osteoblastoma-like variant of osteosarcoma. Bertoni et al\(^41\) reported a series of 11 patients with osteoblastoma-like osteosarcoma and noted that the lesions histologi-
cally resembled osteoblastoma, with peripheral infiltration into surrounding tissues. The tumors had characteristic features of low-grade osteosarcoma, with moderate cellular atypia under high-power microscopic examination.

**Biology and Pathophysiology**

The unique pathophysiologic relationship between the presence of nerve endings and vessels, and increased production of prostaglandins in osteoid osteoma, has not been similarly documented for osteoblastoma. Local pain associated with osteoblastoma is most likely caused by local expansion of the tumor and the pressure of its mass on surrounding structures.40

Other common manifestations of osteoblastoma are local swelling and tenderness, especially when the tumor is near the surface. Gait disturbance can be seen in children with lower extremity involvement. Because of its size and predilection for the vertebral column, osteoblastoma frequently presents with neurologic symptoms resulting from spinal cord or nerve root compression, such as numbness, tingling, radicular pain, paresthesias, and paraparesis. Scoliosis and torticollis may be observed secondary to muscle spasm associated with osteoblastoma.27,17,38,40

In a series of 306 patients with osteoblastoma, Lucas et al37 found progressive pain to be the most frequent complaint (87%). Local swelling, tenderness, warmth, and gait disturbance were also mentioned frequently. The average duration of these complaints prior to diagnosis was 2 years. Ten patients presented with neurologic complaints secondary to spinal tumors, and four had scoliosis.

**Clinical Features**

Clinically, osteoblastoma does not exhibit the typical presenting symptoms and signs seen in patients with osteoid osteoma. In patients with osteoblastoma, the most common presenting complaint is pain, which is usually described as dull, aching, and often progressive in intensity. Typically, pain does not respond dramatically to NSAIDs and is not generally most severe at night.38,40

**Diagnostic Imaging**

Because of the nonspecific signs and symptoms associated with osteoblastoma, radiologic studies are imperative to establish the diagnosis. In sporadic cases, the patient may be

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**Figure 7**

Histologic appearance of the microscopic features of osteoblastoma. A, Low-magnification image demonstrating a well-circumscribed tumor with a thick fibrous capsule (arrows) and irregular woven bony trabeculae with differing degrees of mineralization (box) embedded in a loose fibrovascular stroma (asterisks) (hematoxylin-eosin). B, High-magnification image demonstrating plump and mildly pleomorphic spindle cells (arrows) and osteoclastic giant cells (asterisks) (hematoxylin-eosin). C, High-magnification image demonstrating bony trabeculae lined by a single layer of osteoblasts (arrow) (hematoxylin-eosin). (Adapted with permission from Chakrapani SD, Grim K, Kaimakchiev V, Anderson JC: Osteoblastoma of the spine with discordant magnetic resonance imaging and computed tomography imaging features in a child. Spine [Phila Pa 1976] 2008;33[25]:E968-E970.)

**Figure 8**

pain-free or asymptomatic, and the diagnosis can be obtained incidentally.1,42

**Radiography**
The radiographic appearance of osteoblastoma depends on the location and maturity of the tumor.27,37 In general, these tumors appear as irregularly shaped radiolucent lesions surrounded by a thin shell of reactive bone.40 The interior of the tumor may show various degrees of ossification, which tends to increase as the lesion matures1,37,39. Compared with osteoid osteoma, osteoblastoma lesions appear larger in diameter, with less reactive bone sclerosis and more cortical expansion (Figure 9, A).

Plain radiographs may be useful in establishing the diagnosis. However, radiographic appearance is usually not distinctive, and additional imaging studies are required for diagnostic accuracy. Based solely on radiographs, Lucas et al17 reported diagnostic accuracy of 43% in 116 patients with appendicular osteoblastomas and 64% in 66 patients with vertebral osteoblastomas. The authors noted that the radiographic appearance was not completely reliable in distinguishing osteoblastoma from osteosarcoma; lesions were misinterpreted as malignant in approximately 15% of cases. The radiographs of 17% to 33% of patients were considered to be indeterminate.

**Scintigraphy**
Osteoblastomas exhibit marked uptake of bone-seeking radionuclide on bone scintigraphy because of increased osteoblastic activity within the tumor (Figure 9, B). Scintigraphy is sensitive to but not specific for osteoblastoma. It may be helpful in localizing the lesion, as in cases of osteoid osteoma. However, it may be that the larger size of the tumor may make it relatively easier to localize the lesion without the need for bone scintigraphy.

**CT**
CT is the imaging method of choice for osteoblastoma.6,43 It can provide the most specific information about the location, size, extent, and nature of the tumor. Areas of mineralization within the lesion as well as cortical destruction and soft-tissue extension can be well-delineated on CT sections.1,18,42 Characteristically, the lesion tends to demonstrate areas of mineralization centrally, expansile bone remodeling, and signs of reactive sclerosis toward the periphery with a thin marginal bone shell6 (Figure 10).

**MRI**
The usefulness of MRI in the diagnosis of osteoblastoma is questionable. The features are nonspecific and sometimes confusing, and there may be an overestimation of the extent and nature of the tumor as a result of increased local inflammatory reaction and extensive marrow edema.6,42-44 However, MRI may be a valuable tool in cases in which the tumor has extensive effects on the spinal canal and cord. MRI also may be useful in the evaluation of intra- and extraosseous reactive changes and the presence of soft-tissue infiltration associated with osteoblastoma. The lesion generally appears as low or intermediate signal density on T1-weighted images and intermediate to high signal density on T2-weighted images6,38,45 (Figure 11).

**Management and Prognosis**
Osteoblastoma must be managed surgically because of its potential for aggressive behavior and bone destruction (Figure 4). The selection of surgical procedure depends largely on the location and aggressiveness of the tumor. Intralesional curettage and en bloc resection are the most
commonly performed surgical procedures.

**Intralesional Curettage**

Intralesional curettage is sufficient in most cases. To minimize recurrence, curettage should be extended to normal bone with a high-speed burr. Cryotherapy and chemical cauterization with phenol are valuable adjuncts, and cementation or bone grafting should be performed as indicated. Although curettage may have lower morbidity than en bloc resection, this procedure may leave behind microscopic tumor, which is a source for recurrence.

**En Bloc Resection**

En bloc resection is an effective surgical approach for locally aggressive and large tumors in appropriate cases. In particular, recurrent lesions following intralesional curettage are most successfully managed with en bloc resection. This is also the preferred method for lesions located in expendable bones such as the ribs, clavicle, and fibula.

Berry et al. reported on 99 osteoblastoma patients treated surgically with curettage and bone grafting or en bloc resection. They noted that “23% of the patients treated with curettage required further surgery for recurrence compared to 14% of the patients who underwent en bloc resection.” Five patients had two or more subsequent recurrences. Patients who required more than one reoperation were ultimately treated definitively with en bloc resection.

**Summary**

Osteoid osteoma and osteoblastoma are distinct benign bone-producing tumors with certain similar features. It is crucial to recognize the typical clinical presentation and imaging findings of both types tumor to prevent confusion and misdiagnosis. The differences in management are considerable. Nonsurgical management with NSAIDs may be a valid option for osteoid osteoma. Surgical management is considered when nonsurgical methods fail or are not feasible for or desirable by the pa-
Osteoid Osteoma and Osteoblastoma


Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 15, 23, and 24 are level III studies. References 2, 7, 11, 13, 14, 16-18, 20-22, 28-31, 33, 34, and 37-41 are level IV studies. References 1, 6, 8, 19, 27, 32, 43, and 45 are level V expert opinion. References 3, 5, 9, 10, 12, 25, 26, 35, 36, 42, and 44 are case reports.

References printed in bold type indicate references published within the past 5 years.


