Paget's Disease of Bone: Pathophysiology, Diagnosis, and Management

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Abstract

Paget's disease of bone is a common geriatric disorder of skeletal remodeling, which may have a viral etiology. Safe and effective treatments are now available for associated complications of symptomatic involvement. The orthopaedic surgeon should have a fundamental understanding of the complications of Paget's disease and should be familiar with the indications for treatment, as well as available medical and surgical therapies.

J Am Acad Orthop Surg 1995;3:336-344

The orthopaedic surgeon is often involved with family practitioners, general internists, endocrinologists, and rheumatologists in the diagnosis and care of patients who have Paget's disease of bone. The orthopaedic surgeon may be recruited to render a definitive diagnosis, to participate in patient education, to prescribe medical or physical therapy, or to perform a surgical procedure. Accordingly, the orthopaedic surgeon should be familiar with all aspects of diagnosis and available medical and surgical treatments for patients who have Paget's disease. This article will review the epidemiology, pathology, etiology, pathophysiology, diagnosis, and medical management of Paget's disease of bone.

Epidemiology

Paget's disease of bone, or osteitis deformans, is a common geriatric problem, occurring in 3% to 4% of the population over 50 years of age.[1] It is slightly more common in men.[2] The prevalence has been reported to be 10% to 15% by the ninth decade of life. The disorder is rarely recognized before age 40.

Several members of a family may manifest Paget's disease. As many as 25% of patients have one or more family members who also have the disorder. The cumulative incidence of Paget's disease to age 90 is approximately four times greater in relatives of patients with the condition than in relatives of control subjects. In many families, genetic susceptibility is also suggested by the finding of a linkage between the HLA-DQW1 haplotype and the presence of Paget's disease. These data do not establish a genetic basis for the disease, but rather define familial characteristics that have been observed in a large number of affected patients.[3]

There is a striking geographic distribution of Paget's disease. The disease is most prevalent in England, western Europe, the United States, Australia, and New Zealand, but is uncommon in Scandinavia, China, Japan, and India.[4]

Pathology

Paget's disease is a focal disorder of skeletal remodeling in which the primary cellular abnormality is an increase in osteoclastic resorption of bone (Fig. 1). Increased bone resorption leads to a compensatory increase in bone formation. The overall rate of bone remodeling is accelerated, resulting in a predominance of highly vascular bone that is structurally weak and prone to deformities and pathologic fractures.[1,2,4,5]

Pagetic osteoclasts are more numerous and larger than normal osteoclasts and may contain as many as 100 nuclei per cell. These bone-resorbing cells often contain nuclear and cytoplasmic inclusions, which

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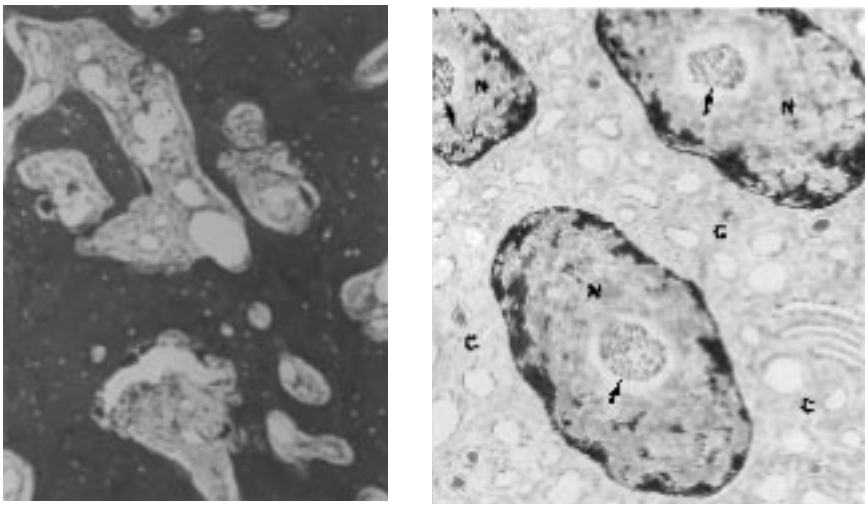

A B

Fig. 1 Light- and electron-microscopic findings in Paget's disease. **A,** Section of bone shows intense osteoclastic and osteoblastic activity and mosaic of lamellar bone. **B,** Electron-microscopic view of multinucleated osteoclast with nuclear inclusions that may be viruses (arrows). N = nuclei; C = cytoplasm. (Reproduced with permission from Netter FH: *The CIBA Collection of Medical Illustrations.* Summit, NJ: CIBA-Geigy Corp, 1987, vol 8, pt I, p 238.)

resemble nucleocapsids of the Paramyxoviridae family of viruses.[6]

The pathologic process in Paget's disease may be divided into active and inactive phases. Early in the active phase, intense osteoclastic bone resorption prevails (lytic phase). Later, compensatory bone formation is apparent (mixed phase). Very late in the active phase, osteoblastic bone formation predominates (sclerotic phase). Occasionally, the disease may reach an inactive phase in which a sclerotic lesion may remain in the absence of excessive bone-cell activity. In such a lesion, the adjacent marrow consists predominantly of fat cells with few areas of hematopoietic or fibrovascular elements.[5]

Pagetic bone is usually lamellar and fully mineralized. In very active lesions, however, immature woven bone may be present in regions of poorly mineralized osteoid. The affected bone has a characteristic mosaic pattern with irregularly shaped areas of lamellar bone and an erratic pattern of cement lines. This mosaic pattern is a consequence of accelerated bone resorption and formation.[5]

Etiology

A number of hypotheses have been proposed to explain the genesis of Paget's disease. The most likely cause is a slow viral infection of bone,[7] perhaps with an underlying familial predisposition.

Immunohistologic studies of pagetic osteoclasts have revealed the antigens of paramyxoviruses, particularly the measles and respiratory syncytial viruses. Further support of a viral etiology is provided by the finding of the mRNA of paramyxoviruses in some pathologic material. Although an infectious virus has not yet been isolated from long-term cultures of pagetic bone cells, this does not eliminate a viral etiology, because the putative virus is highly defective[4,6] and therefore no longer capable of being cultured.

Current data suggest that Paget's disease may be caused by hematogenous infection with one of several possible paramyxoviruses and that following an acute viremia, osteoclasts or their precursors become chronically infected. According to this hypothesis, the virus mutates rapidly and loses its infectivity but is able to stimulate osteoclast proliferation and activity, possibly by cytokine production. This may induce dramatic local osseous effects.[7,8] Various familial and genetic factors may be involved in susceptibility and immunologic reactivity to the putative infectious agent, which would likely influence the clinical expression of the disease.[3,5,6,9]

Clinical Manifestations and Complications

Paget's disease may affect any bone and may be monostotic or polyostotic. Clinical manifestations vary, depending on the sites and severity of lesions (Tables 1 and 2). Many individuals with radiologic evidence of the disease have no symptoms and may not require treatment.[1,2,4] In fact, because Paget's disease is usually asymptomatic, it most often is detected incidentally on radiographs obtained for other purposes or is suggested by the unexpected elevation of serum alkaline phosphatase activity on a routine serum chemistry screening panel.

In symptomatic patients, bone pain can be mild to severe and is usually unrelated to physical activity. Acute pain may develop as a consequence of pathologic fractures of affected bone. A variety of other local and general signs and symptoms may also occur.

The characteristic bone enlargement that occurs with Paget's disease may cause spinal stenosis,

Table 1
Common Diagnostic Features of Paget's Disease of Bone

History
Rare before 40 years of age
Insidious onset of symptoms (pain, deformity, headache)
Often asymptomatic
Physical findings
Bone deformity and enlargement
Increased heat (due to increased blood flow) over affected bone
Bone tenderness
Asymmetric limb involvement
Associated neurologic involvement
Associated pagetic arthritis
Biochemical findings
Increased serum alkaline phosphatase level
Increased urinary excretion of pyridinoline cross-links
Radiologic findings
Metaphyseal involvement
Sclerotic and lytic changes in same bone
Coarse, thick trabeculae
Enlargement of bone
Associated arthritic changes (late)
Increased radionuclide uptake on technetium-99m methylene diphosphonate bone scan
Bone pathology findings (often from surgical specimens)
Disordered remodeling
Increased osteoclastic and osteoblastic activity
Giant osteoclasts
Mosaic pattern of lamellar bone
Presence of woven bone
Virus-like inclusions in nuclei and cytoplasm of osteoclasts on electron microscopy

resulting in spinal radiculopathy or cauda equina syndrome.[10] Increased blood flow to the highly vascular pagetic bone has been thought to provoke the "steal syndrome." In this situation, blood is shunted away from the neural elements, exacerbating the neurologic symptoms and signs accompanying the stenosis.[10]

The function of the cranial nerves may also be affected. Pagetic involvement of the temporal bone is a common cause of conductive hearing loss in this condition. Less commonly, involvement of the cochlea results in mixed sensory and conductive deafness. Extreme thickening and enlargement of the skull may result in platybasia and, in rare instances, impaired cerebrospinal fluid flow and hydrocephalus.[9]

Painful fissure fractures, pseudofractures (transverse radiolucent areas on the convex side of a long bone), and complete pathologic fractures (transverse "chalk-stick" fractures) may occur in areas of high stress, particularly in the weight-bearing bones of the lower limbs. Fracture healing may be impaired, resulting in delayed union or nonunion. This complication is most common with fractures of the femur.[1,4,5]

Articular disorders commonly associated with Paget's disease of bone are degenerative arthritis, calcific periarthritis, and gout.[11,12] Degenerative arthritis in joints contiguous with pagetic bone is likely to occur because of accelerated remodeling of the affected bone, which results in juxta-articular bone enlargement, abnormal joint biomechanics, and altered subchondral support. When the hip is affected, acetabular protrusion along with medial joint-space narrowing may result.[1,2,4] The association of gout with Paget's disease may be secondary to hyperuricemia due to accelerated nucleic acid turnover in subjacent bone.[11,12]

Metabolic complications of Paget's disease are uncommon but include hypercalciuria and hypercalcemia.[1,4] These complications are seen in only the most severely involved (polyostotic Paget's disease) and immobilized patients. The association of mild hyperparathyroidism and Paget's disease has been reported, but its significance is unclear and may be coincidental.[1,4]

The most serious complication of Paget's disease is the development of a malignant bone tumor.[13] Osteosarcomas, chondrosarcomas, fibrosarcomas, and tumors of mixed histologic characteristics may develop, almost always in a preexisting pagetic lesion. Primary giant cell tumors and secondary metastatic spread of carcinoma to pagetic bone are also quite common.

Paget's sarcoma, a rare complication, occurs in fewer than 1% of affected patients and is rare before 70 years of age.[13] A marked and sustained increase in pain in an area of long-standing Paget's disease in an elderly patient suggests this serious sequela. Other manifestations are night pain and radiographic evidence of bone destruction. Serum alkaline phosphatase activity may be unaltered and thus is not a useful test. Rapid worsening of bone pain or deformity should indicate the need for radiologic evaluation, followed by bone biopsy if suspicion of a tumor remains. Magnetic resonance imaging and computed tomography are particularly helpful in delineating the presence of bone tumors. Despite recent advances in the treatment of malignant bone tumors, the prognosis remains very poor in patients who have Paget's disease.[13] There are currently no data to support the preventive effects of chronic suppressive medical therapy on the risk of pagetic sarcoma.

Diagnostic Evaluation

The initial evaluation of a patient with Paget's disease should include a complete history and physical examination, a radionuclide bone

Table 2
Manifestations of Paget's Disease of Bone

Nonskeletal
Neurologic
Hearing loss and, less commonly, other cranial nerve deficits
Spinal-cord or nerve-root compression
Cardiovascular
Increased cardiac output due to increased bone vascularity
Possible high-output congestive heart failure
Metabolic
Hypercalcemia, hypercalciuria, and urolithiasis in immobilized patients
Hyperuricemia and gout
Primary hyperparathyroidism (possibly coincidental)
Dental
Poor occlusion
Tooth loosening and loss
Oncologic
Pagetic osteosarcoma
Pagetic chondrosarcoma
Pagetic fibrosarcoma
Tumors of mixed histologic characteristics
Primary giant cell tumors
Skeletal
Axial skeleton
Back pain due to vertebral body involvement
Osteoarthritis of facet joints
Diskogenic disease secondary to vertebral involvement and alteration of nourishment to disk
Spinal stenosis (due to vertebral body enlargement)
Spinal-artery steal syndrome with paraparesis and/or paraplegia
Compression fractures of vertebral bodies
Neoplastic degeneration
Appendicular skeleton
Bone pain and tenderness
Progressive deformity
Secondary osteoarthritis
Pathologic fractures with high rate of delayed union, nonunion, or malunion
Neoplastic degeneration

scan, appropriate radiographs, and baseline laboratory tests (serum alkaline phosphatase, calcium, phosphorus, and albumin determinations).[2,4]

Radiologic Studies

Radioisotope studies should be performed to evaluate the metabolic activity of a pagetic lesion, as well as to assess the total skeletal involvement in a patient who has focal evidence of the disease (Fig. 2). A bone scan should be performed when there is a question about the metabolic activity of a specific region of the skeleton or when it is desirable to document the full extent of the disease. A complete radiographic examination should be obtained, including all skeletal sites that demonstrate an increase in radionuclide uptake.[2,4]

Technetium-labeled bisphosphonates are most commonly used in radioisotope scanning. Localization of the bone-seeking agent Tc-99m methylene diphosphonate is dependent on the relative vascularity of the bone and the extent of hydroxyapatite crystal surface available for binding of the compound. Bone scans are far more sensitive than radiographs in detecting occult pagetic lesions, although inactive sclerotic lesions may be missed.

The diagnosis of Paget's disease is usually established on the basis of the characteristic radiographic appearance of the lesions. The earliest lesions of Paget's disease are osteolytic and are most readily observed in the skull and long bones (Fig. 3). In the skull, discrete oval or round areas of osteolysis are termed osteoporosis circumscripta. In the limbs, the disease usually begins as localized metaphyseal involvement. The osteolytic lesion usually has a V or arrowhead shape at its advancing edge. Lesions have been observed to progress slowly toward the opposite end of the affected long bone at a rate of approximately 1 cm/yr in untreated patients.[2,4]

As the osteolytic process progresses to involve much of the long bone or skull, the more familiar osteosclerotic lesions of Paget's disease replace the earlier osteolytic regions. Over a period of years to decades, the bone becomes chaotic in structure and thickened. The overall bone size may increase remarkably.

In the lower limbs, bowing, pseudofractures, and complete transverse pathologic fractures are common. The latter chalk-stick fractures (Fig. 4) can also be seen in osteomalacia or fibrous dysplasia. Subchondral bone involvement in the metaphysis on one side of a diarthrodial joint may lead to joint incongruity and subsequent arthritis.

In patients with back pain, magnetic resonance imaging and computed tomography have been

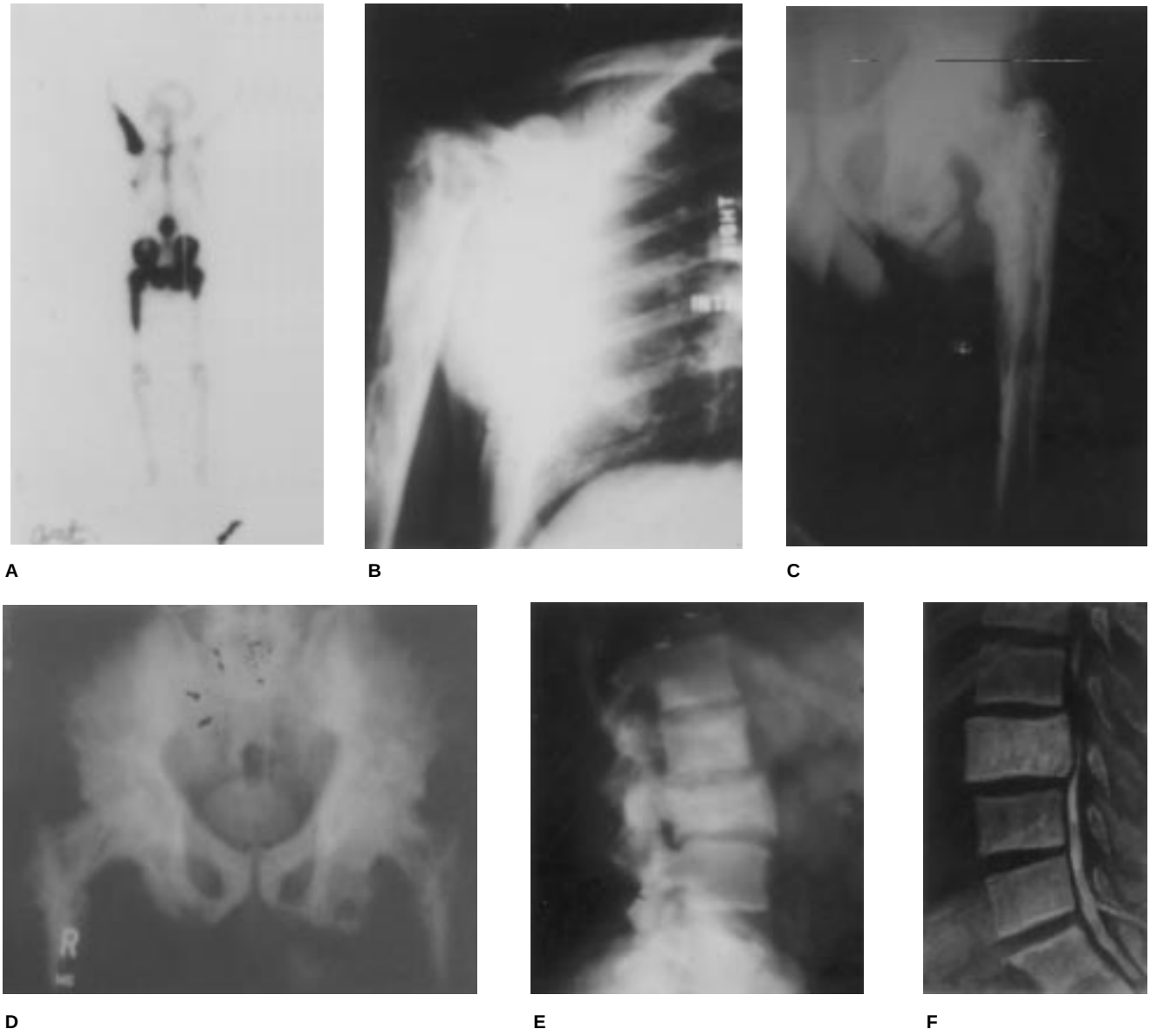

Fig. 2 Images of a patient with Paget's disease. **A,** Bone scan shows areas of increased isotope uptake. **B,** Proximal humerus appears thickened and coarsely trabeculated, with patchy rarefaction. **C,** Proximal femur shows typical manifestations of the disease, including osteosclerotic and osteolytic areas, bone enlargement, and metaphyseal involvement with coarse trabeculation. **D,** Disease involvement can be seen throughout the pelvis and the proximal femurs, with involvement of the hip joints. **E,** The body of L3 appears enlarged, with increased density. **F,** Myelogram shows obstruction of the vertebral canal at L3. (Reproduced with permission from Netter FH: *The CIBA Collection of Medical Illustrations.* Summit, NJ: CIBA-Geigy Corp, 1987, vol 8, pt I, p 237.)

particularly useful in defining the extent of degenerative arthritis, spinal stenosis, and nerve-root impingement.[10] An increase in the size of a vertebral body is one of the radiographic hallmarks of Paget's disease of the spine, which can be particularly helpful in differentiating Paget's disease from lymphoma or metastatic carcinoma.

Biochemistry

The measurement of serum alkaline phosphatase activity is the most useful biochemical test for Paget's disease. The activity of this enzyme, which is located in the plasma membrane of osteoblasts, reflects the number and functional state of osteoblasts in patients who have bone disease. In Paget's disease, the level of alkaline

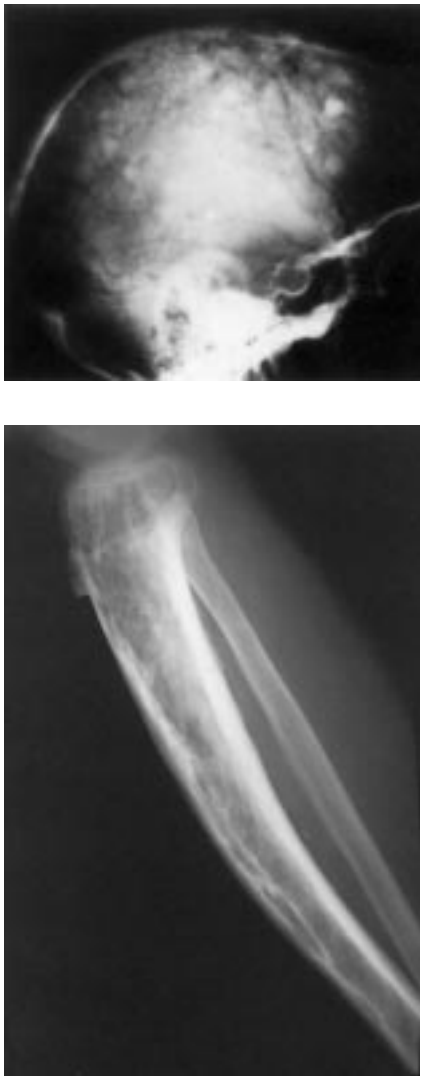

Fig. 3 Characteristic early lesions of Paget's disease. **Top,** Lateral radiograph shows patchy density of the skull, with areas of osteopenia (osteoporosis circumscripta cranii). **Bottom,** Radiographic findings in the tibia include thickening, bowing, coarse trabeculation, and an advancing radiolucent wedge. (Reproduced with permission from Netter FH: *The CIBA Collection of Medical Illustrations.* Summit, NJ: CIBA-Geigy Corp, 1987, vol 8, pt I, p 236.)

phosphatase activity correlates roughly with the extent of skeletal involvement as established by radioisotope bone scanning. Serial alkaline phosphatase determinations generally provide a useful, simple, and inexpensive biochemical index of disease activity. In selected patients, new assays specific for bone alkaline phosphatase may be helpful.

Urinary excretion of pyridinoline cross-links, a biochemical index of bone-matrix collagen resorption, is measured over 24 hours. As with serum alkaline phosphatase activity, urinary excretion of pyridinoline cross-links correlates well with the extent of pagetic involvement.[14] Because of the expense and inconvenience, this test is not generally used routinely in the assessment or follow-up of a patient with Paget's disease.[1,2,4]

Due to tight metabolic coupling between bone resorption and bone formation, calcium levels in the serum and urine are usually normal except when there is concurrent generalized immobilization, hyperthyroidism, hyperparathyroidism, or malignancy.[1,2,4]

General Diagnostic and Treatment Considerations

Not everyone with Paget's disease will require treatment. In many cases, the symptoms that cause the patient to seek care are due to an associated disorder. Therefore, a careful consideration of the symptoms and the physical and radiographic findings is necessary to determine whether treatment of Paget's disease is indicated.

Nonsteroidal anti-inflammatory medications and aspirin continue to have an important role in treating the arthritis associated with joint destruction in Paget's disease. Routinely, patients with end-stage Paget's disease have low alkaline phosphatase levels, limited disease activity, and moderate joint degeneration, which can be symptomatically controlled with non-steroidal anti-inflammatory agents. The direct analgesic effects of these medications are also important, but this benefit is commonly overlooked in practice. Narcotic agents should be avoided, if possible, particularly in the older population.

A cane can be a very important therapeutic device for patients with Paget's disease of the lower limbs. Because of the benefits of increased stability, prevention of falls, and load-sharing capability, the use of a cane makes good sense and should be standard treatment for the elderly patient.

The most important indications for treatment of Paget's disease include bone pain, neurologic involvement, high-output cardiac failure, prevention of fracture or skeletal deformity in young patients

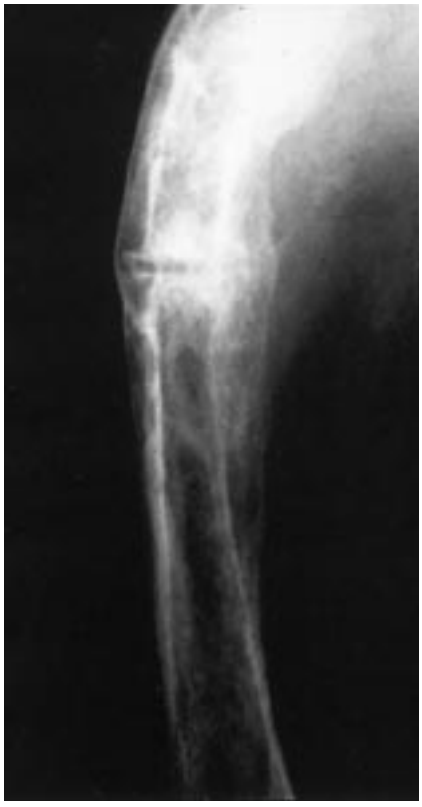

Fig. 4 Healing chalk-stick fracture. (Reproduced with permission from Netter FH: *The CIBA Collection of Medical Illustrations.* Summit, NJ: CIBA-Geigy Corp, 1987, vol 8, pt I, p 236.)

with active disease, and preparation for orthopaedic surgery (Table 3).[4,15]

Before considering the use of specific therapy for Paget's disease, a thorough explanation of the disease should be offered to the patient. Treatments should be discussed in the context of arresting progression of the disease. In all discussions and planning, relief of pain and restoration of function should be emphasized. The need for long-term follow-up should also be stressed.

All patients should be reexamined at least annually, and the alkaline phosphatase level should be determined at these visits. Careful questioning, examination, and correlation with changes in alkaline phosphatase levels will often reveal subtle symptoms and signs of disease activity or progression that might be amenable to therapy. Radiographs may be obtained periodically, as dictated by symptoms. Repeat bone scans are not obtained routinely unless bone pain occurs at a new site or new symptoms arise.

The home environment of the patient should be discussed with the goal of preventing accidents that could lead to pathologic fractures. In patients who have auditory deficits due to Paget's disease, attention to auditory acuity may be helpful to detect subtle changes.[9] Patients with maxillary or mandibular involvement should be referred for dental evaluation.[9]

The Paget Foundation (200 Varick Street, New York, NY 10014) can be helpful in providing patient information on the disease.

Table 3
Indications for Drug Therapy in Paget's Disease

Bone pain
Preparation for orthopaedic surgery
Treatment of medical complications, including hearing loss, spinal stenosis with nerve dysfunction, and high-output congestive heart failure
Prevention of fracture or skeletal deformity in patients with rapidly progressive osteolytic lesions or in young patients with active disease

Medical Treatment

The two major types of therapeutic agents currently used in the treatment of symptomatic Paget's disease are calcitonin and the bisphosphonates.[1,15] The treatment schedules and special characteristics of each agent are presented in Table 4. The observed benefits of long-term calcitonin therapy include relief of bone pain, a reduction of increased cardiac output, reversal of certain neurologic deficits, stabilization of hearing loss, healing of osteolytic lesions, and reduction in complications of orthopaedic surgery, such as excessive bleeding.

Calcitonin is a safe and highly effective treatment for Paget's disease. Most patients are able to self-administer daily subcutaneous injections. Side effects (usually minor) occur in about 20% of patients treated with either salmon calcitonin or human calcitonin. These include nausea, facial flushing, and polyuria. In approximately 20% of patients, resistance to chronic salmon calcitonin therapy develops after a successful initial treatment period. If long-term treatment with salmon calcitonin or human calcitonin is discontinued, exacerbation of biochemical abnormalities and symptoms may occur within 1 year.[15]

Another class of drugs useful in treating Paget's disease is the bisphosphonates (formerly diphosphonates). These compounds are pyrophosphate analogues, which are potent inhibitors of bone resorption. They bind to the hydroxyapatite crystals in bone and may remain in bone for a prolonged time after the discontinuation of treatment.[15]

Etidronate disodium was the original bisphosphonate approved for routine clinical use. The advantage of this drug is that it can be used orally. After absorption, the drug localizes to bone or is excreted unchanged in the urine. The recommended dose of etidronate (5 mg/kg of body weight daily for 6 months) produces suppression of disease activity and symptoms similar to that which occurs with calcitonin. Because absorption of the drug is poor and variable, it should be taken once daily on an empty stomach at least 2 hours remote from any other food; the medication may be taken with a glass of water. Etidronate should not be used in the treatment of patients with osteolytic pagetic lesions, for whom calcitonin is preferable.

Side effects appear to be less common with etidronate than with calcitonin; loose bowel movements and nausea may occur but are infrequent. Hyperphosphatemia is sometimes observed, particularly if higher-than-recommended doses are given; the dose should be reduced if this is found. High doses or long-term uninterrupted use also produce a mineralization defect and might predispose to pathologic fractures.[1,2,15]

After the recommended 6-month treatment course, biochemical and symptomatic remissions may persist for months or, occasionally, for years. In most patients, biochemical indices return toward pretreatment levels within a year. When symptoms recur, repeat 6-month treatment courses often will produce biochemical and symptomatic improvement similar to that achieved with the initial treatment course. However,

Table 4
Drug Treatment of Paget's Disease

Drug	Dosage	Special Characteristics
Salmon calcitonin	50-100 international units subcutaneously daily; after symptomatic improvement, may reduce to 3 times weekly	Anti–salmon calcitonin antibodies develop in 60% of patients; clinical resistance in >20%
Human calcitonin	0.5 mg subcutaneously daily; after symptomatic improvement, may reduce to 3 times weekly	Effective in salmon calcitonin–resistant patients with high antibody titers
Etidronate disodium	5 mg/kg of body weight orally for 6 months; may repeat after recurrence of symptoms or biochemical exacerbation, but not sooner than 6 months	Remission for years may occur after 6 months of therapy; some patients experience transient increased bone pain; osteolytic lesions rarely heal; osteomalacia occurs at high doses
Pamidronate disodium	30 mg intravenously slowly over 3½ to 4 hr; repeat daily for 3 consecutive days; repeat as needed; patients with more severe disease may need 60 mg monthly or quarterly for variable periods	New bisphosphonate with potent antiresorption effects; transient fever (< 24 hr) is common side effect

resistance to repeated courses of treatment is common.

Second-generation bisphosphonates that are more potent than etidronate have been developed. These agents do not impair mineralization. The most widely evaluated is pamidronate disodium,[16] which has recently been approved by the Food and Drug Administration for use in Paget's disease. The medication is administered intravenously because of its poor gastrointestinal absorption. The approved treatment regimen is 30 mg/day by slow (duration, 3½ to 4 hours) intravenous infusion for 3 consecutive days; the regimen may be repeated as needed. The drug can also be administered in a 60-mg single dose or in a weekly, monthly, or quarterly regimen, depending on the severity of the disease and the response of the patient. A brief postinfusion fever or acute pain flare may occur. Short courses of treatment with pamidronate can produce long-term remissions of the disease. Oral preparations of some of the newer bisphosphonates will likely be available in the near future. Thus, it is likely that new-generation bisphosphonates will become the most commonly used and effective therapy for Paget's disease.[16]

Surgery

Some indications for surgical intervention in Paget's disease include femoral fractures and severe arthritis that is refractory to medical treatment. Malalignment of the major weight-bearing bones or impending fractures may be treated with orthoses or with surgery. Preoperative medical treatment with bisphosphonates or calcitonin decreases intraoperative bleeding. Spinal decompression for spinal stenosis is an important consideration, but there are meager data on the management of this complication. Spinal surgery should be avoided until all nonsurgical options have been diligently tried. Assiduous attention must be paid to the surgical approach in order to preserve spinal stability.

Surgical intervention in Paget's disease is most often sought when degenerative arthritis of the hip or knee produces severe pain on weight-bearing and impaired mobility.[17,18] Anti-inflammatory agents usually produce little relief of symptoms in this setting. Diagnostic intra-articular injections with a local anesthetic often confirm that the pain is primarily articular, rather than osseous. Total hip replacement is highly effective in relieving hip pain and restoring mobility.[18] Tibial osteotomy is equally effective in relieving knee pain in patients who have severe tib-

ial bowing if the associated articular degeneration is not too advanced.[19]

Before any operative orthopaedic procedure is performed, it is desirable, if possible, to reduce disease activity with drug therapy in order to prevent excessive blood loss. A reduction in serum alkaline phosphatase activity to approximately 50% of pretreatment levels is probably adequate preoperative control. In elective cases, it is therefore desirable to begin antipagetic medication at least 6 weeks before surgery.[2] When osteotomy followed by bone healing is planned, calcitonin and pamidronate are the drugs of choice.

When total joint replacement is performed, long-term suppression of disease activity through use of calcitonin or pamidronate may be desirable to diminish excessive bone-remodeling activity and prevent loosening of prosthetic components.[17,18] In all circumstances requiring surgery, the patient must be aware that delayed bone healing may occur and that a lengthy rehabilitation program may be necessary.

Summary

Paget's disease is a common condition in the elderly, and its complications are a common source of skeletal morbidity and symptoms. Orthopaedic physicians should be aware of the disorder and its myriad manifestations. Although a cure is currently unavailable, a wide array of medical and surgical therapeutic options offer much hope to those afflicted by symptomatic complications.

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