Editorial

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Editorial Note on Chronic Osteomyelitis

Neil Baker

Department of Medicine, Molecular Biology Institute, I-Shou University, Kaohsiung, Taiwan

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***Corresponding author:** Neil Baker

Department of Medicine, Molecular Biology Institute, I-Shou University, Kaohsiung, Taiwan.

NeilBaker443@gmail.com

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Editorial

Chronic osteomyelitis is distinguished by an increase in inflammatory neutrophils, lymphocytes, and plasma cells, as well as the presence of sequestrum (infected dead bone) and involucrum (a surrounding formation of new bone). Pus frequently perforates the periosteum and forms a sinus tract to the skin in acute osteomyelitis. As the sinus tract heals, the epithelium of the sinus tract may become entrapped within the bone, forming inclusion cysts or, eventually, squamous carcinoma.

Epithelial inclusions are also frequently seen as bone marrow biopsies' artefacts. When a biopsy needle is pushed through the skin overlying the biopsy site, fragments of skin or other dermal or subcutaneous structures can end up adjacent to or appearing to be within the bone marrow space in histologic sections. Floaters from other biopsies may appear in sections and should be suspected if there is space between the unexpected tissue and the bone marrow biopsy. If this is suspected, the procedure should be repeated.

Patients with a history of normal skeletal development but skeletal pain or fracture, as well as radiologic evidence of osteopenia, may be suffering from metabolic bone disease. Active osteoporosis (with accelerated bone turnover) results in increased osteoid formation and a higher proportion (>20%) of trabeculae with normal-width osteoid seams. There are more than four collagen lamellae layers present, and bone surfaces have plump osteoblasts. There are also more osteoclasts (>1 to 2 per section and/or clustered). Peritrabecular fibrous tissue (osteitis fibrosa) similar to hyperparathyroidism may be observed. Inactive osteoporosis (low turnover) is characterised by thin osteoid seams, flattened osteoblasts, and decreased osteoclasts. There is bone formation and resorption, but there is less bone tissue loss overall.

Calcification abnormalities include osteomalacia and rickets (vitamin D deficiency). Osteomalacia is difficult to diagnose histologically and may require fluorescence examination after tetracycline administration; positive results show a decrease in fluorescence deposition. Rickets causes uncalcified masses of cartilage in a child's growth plate. Hyperparathyroidism, whether primary (due to a parathyroid adenoma) or secondary (due to renal failure), causes increased osteoclastic and osteoblastic activity, as well as peritrabecular fibrosis, also known as osteitis fibrosa. Scurvy (vitamin C deficiency) impairs the formation of osteoid due to abnormal collagen transformation. Calcified cartilage is seen with increased density at the growth plate on radiographs.

Chronic osteomyelitis has become much less common in developed countries as a result of antimicrobial therapy and improved diagnostic techniques. The major factors now associated with chronic disease are delayed diagnosis, inadequate antimicrobial or surgical therapy, and resistant organisms. Characteristic findings include extensive necrosis, sequestrum formation, and decompression caused by fistulization through the overlying soft tissues. Patients experience varying degrees of local pain and frequently have chronic draining sinus tracts. To achieve resolution, aggressive surgical curettage and long-term antimicrobial therapy are required; however, permanent functional disability and deformity are not uncommon in chronic osteomyelitis. Chronic recurrent multifocal osteomyelitis (CRMO), a rheumatologic condition with a similar clinical picture, is the main differential consideration for chronic bacterial osteomyelitis. However, this disorder is distinguished by multiple recurrent sites of sterile bone inflammation that responds to anti-inflammatory therapy, and radiologic findings do not accurately reflect the severity of chronic disease.

Chronic osteomyelitis frequently stimulates the formation of periosteal new bone, which appears radiographically as a single radiopaque line or a series of radiopaque lines parallel to the surface of the cortical bone (similar to onion skin). The radiolucent strip that separates this new bone from the outer cortical bone surface may eventually be filled in with granular sclerotic bone. When this happens, the original cortex may be impossible to identify, making it difficult to determine whether the new bone is derived from the periosteum. After a long period of time, the outer contour of the mandible may be altered, assuming an abnormal shape and the girth of the mandible may be significantly

larger than on the unaffected side. External resorption of tooth roots may occur, and the lamina dura may become less visible as it blends with the surrounding granular sclerotic bone.

When a tooth becomes non-vital, the periodontal ligament space in the apical region usually expands. In patients with advanced chronic osteomyelitis, the disease may gradually spread to the mandibular condyle and into the joint, resulting in septic arthritis. The inner ear and mastoid air cells may be affected in the future. Chronic lesions may form a draining fistula, which appears as a well-defined break in the outer cortex or periosteal new bone.