Abstract. This review reports the natural evolution of benign and malignant lesions of connective tissue derivation that led to the staging system, the system for both benign and malignant lesions, its articulation with surgical treatment and early experience with its use.

Key words: Staging – Natural evolution – Grade – Site – Metastases

The purposes of a staging system for musculoskeletal neoplasms are to: (1) incorporate the significant prognostic factors into a system which describes progressive degrees of risk of local recurrence and distant metastases to which a patient is subject, (2) stratify the stages so they have specific implications for surgical management, and (3) provide guidelines for adjunctive therapies.

Over a number of years, staging systems for various classes of malignant tumors have been developed under the auspices of the American Joint Committee for Cancer Staging and End Results Reporting (AJC). The systems vary among cancers related to the natural course of a particular type of cancer. In 1980, a system for the surgical staging of musculoskeletal sarcoma was proposed, studied, and adopted by the Musculoskeletal Tumor Society [4] and subsequently adopted by the AJC.

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They may slowly attain large size but eventually reach a steady state where they no longer grow. They appear quite responsive to contact inhibition and remain completely encapsulated. They remain intracompartamental and seldom deform the compartmental boundaries of cortical bone, articular cartilage, or dense fascial septae. When palpable in soft tissue, they are often small, movable, non-tender, with little or no significant enlargement on subsequent clinical observation. Radiographic characteristics are lesions that are well marginated by a mature shell of cortical-like reactive bone without deformation or expansion of the encasing bone (Lodwick IA). Angiographic staging studies show little or no increase in isotope uptake and no significant reactive neoangiogenesis about the lesion or intraloskeletal neoangiogenesis. CT scanning shows a homogenous density, well marginated, with no cortical broaching or cross-fascial extension.

The histologic characteristics of the lesion are: (1) low cell-to-matrix ratio; (2) mature, well-differentiated matrices; (3) benign cytologic characteristics (no hyperchromasia, anaplasia, or pleomorphism); (4) encapsulation by mature fibrous tissue or cortical bone; and (5) little or no reactive mesenchymal proliferation, inflammatory infiltrate, or neoangiogenesis about the lesion.

Active benign lesions are mildly symptomatic, discovered because of discomfort and occasionally associated with pathologic fracture or mechanical dysfunction. They grow steadily and continue to enlarge during observation. They appear responsive to contact inhibition but not at normal levels as they can expand by deformation of overlying cortical bone, articular cartilage, or fascial septae. They remain encapsulated and have only a thin layer of filmy areolar tissue forming the reactive zone between the lesion and surrounding normal tissue. When palpable in soft tissue, they are usually small, movable, moderately tender, and grow slowly during clinical observation. Radiographic characteristics of active lesions in bone are well, but irregularly, marginated defects. The margin is a mature cancellous ring rather than a cortical shell, and the inner aspect is often irregular or corrugated giving a septated appearance. Expansion, bulging, or deformation of the combination of overlying cortex/reactive bone (Lodwick IB) is frequently observed.

Staging studies show increased isotope uptake that conforms closely to the limits of the radiographic defect and reactive changes. A thin but discernible rim of reactive neoangiogenesis about the lesion is frequently observed but seldom any significant intraloskeletal neoangiogenesis. CT scanning shows a homogenous density, irregular but intact reactive bone, "expansion" of the overlying cortex, and intracompartamental containment by bone or fascia.

The histologic characteristics of active benign lesions are: (1) a relatively balanced cell-to-matrix ratio with homogenous distribution of the matrix; (2) well-differentiated matrices; (3) benign cytologic characteristics; (4) an intact capsule of mature fibrous tissue and/or cancellous bone; (5) a narrow zone of mesenchymal, inflammatory, and vascular reactive tissue between the capsule and the surrounding normal tissue; (6) resorption of preexisting bone by osteoclasts rather than by neoplastic cells as the mechanism of expansion. Intermittent areas of resorption often produce an irregular, serrated, sometimes corrugated interface between the capsule and the adjacent reactive bone.

Aggressive benign lesions are often symptomatic, discovered because of discomfort and/or a growing mass, and when in a stress-bearing bone associated with a pathologic fracture. When palpable in the soft tissues, they grow rapidly, sometimes alarmingly. These lesions are frequently tender and may have an inflammatory-like appearance. They are little affected by contact inhibition and readily penetrate or permeate the natural barriers to tumor growth: cortical bone, fascial septae, and in some cases, articular cartilage or joint capsules. They penetrate the capsule with finger-like extensions protruding directly into the surrounding zone. The reactive zone is thick, edematous, and often appears inflammatory. These aggressive lesions invade by destroying or resorbing the restraining bone or fascia and permeate into adjacent tissues or compartments rather than expanding by concomitant endosteal resorption and subperiosteal apposition.

When aggressive benign lesions involve unrestrained areas (medullary canal, cancellous bone, advential intermuscular planes within muscle bellies, pararticular tissues), they extend rapidly although usually preceded by a pseudocapsule of reactive tissue. Aggressive soft tissue lesions are usually firm, fixed, tender, and have a rapid growth history. The radiographic features of aggressive benign lesions are a ragged permeative interface with adjacent bone, incomplete attempts at containment by reactive bone, cortical destruction, endosteal buttresses and periosteal Codman's triangles, and rapid soft tissue extension (Lodwick IC). Staging studies often reflect the aggressive nature and be-