

Magnetic Resonance Imaging of Benign Soft Tissue Neoplasms in Adults

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KEYWORDS

- Soft tissue tumor • Benign • MR imaging
- Soft tissue neoplasm

Benign soft tissue lesions outnumber their malignant counterparts by a factor of 100:1.^{1,2} Many of these lesions are small and superficial and do not lead to imaging evaluation or biopsy; so precise estimates are unavailable. Magnetic resonance (MR) imaging is the favored modality for evaluation of soft tissue tumors and tumorlike conditions because of its superior soft tissue contrast, multiplanar imaging capability, and lack of radiation exposure. MR imaging is valuable for lesion detection, diagnosis, and staging.

When planning an MR imaging study for evaluation of a soft tissue lesion, at least 2 orthogonal planes should be obtained. In our experience, lesions are typically best evaluated in the axial plane, and this plane is usually the most familiar to radiologists. The secondary plane of imaging for an anterior or posterior lesion is typically the sagittal plane. Coronal sequences are optimal for evaluation of medial or lateral masses.

T1-weighted (T1W) and T2-weighted (T2W) sequences should be obtained because most soft tissue lesions have been described with their spin echo (SE) T1W and T2W signal characteris-

tics. Fast-spin echo sequences in place of SE sequences can reduce scanning time and patient motion artifacts. Gradient echo sequences can be useful for demonstrating hemosiderin with “blooming” and also are subject to artifact caused by metal, hemorrhage, and air. Short tau inversion recovery and chemical shift-selective fat saturation T2W images increase sensitivity to abnormal tissue containing increased water content. However, in our opinion, these techniques also reduce information concerning various tissue consistencies and should be used in the secondary, not the primary, plane of imaging. The smallest diagnostic field of view is preferable when evaluating these lesions.

The use of intravenous contrast for lesion evaluation is controversial but appropriate in certain circumstances. Gadolinium contrast agents increase the T1W signal intensity of many soft tissue tumors, allowing distinction between tumor and muscle or tumor and edema, but the surrounding area of edema may enhance as well. Information about tumor vascularity is also obtained.^{3,4} Comparing precontrast and postcontrast T1W fat

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Radiol Clin N Am 49 (2011) 1197–1217

doi:10.1016/j.rcl.2011.07.007

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saturation sequences is useful to distinguish true enhancement from a high T1W signal process such as lesion hemorrhage or a proteinaceous fluid collection.

Many investigators have evaluated the use of dynamic enhancement with gadolinium to aid in differentiating benign from malignant soft tissue lesions.^{3,5,6} High soft tissue vascularity and perfusion result in an increased rate of enhancement. Benign lesions usually reveal less enhancement overall and a delayed rate of enhancement.⁷ There is a significant overlap between the rate of enhancement of benign and malignant lesions.⁸ In our opinion and experience, dynamic enhancement does not obviate biopsy of the otherwise indeterminate solid soft tissue mass.

Studies are conflicting regarding the use of tumor margins, homogenous versus heterogeneous signal intensity, and lesion size to distinguish benign from malignant lesions. The most optimistic report suggests the distinction can be made in more than 90% of cases.⁹ Other investigators note that malignant lesions can appear smoothly marginated and homogenous and MR appearance cannot accurately separate benign and malignant processes.^{3,10-14} Only a minority (5%) of soft tissue tumors are larger than 5 cm in diameter, and about 1% of benign lesions are deep.^{15,16} In general, well-defined smooth margins, homogenous signal intensity, and small size are seen with benign lesions. Unless a specific diagnosis can be determined, a lesion should be considered indeterminate and biopsy performed, with an appropriate biopsy path discussed with the orthopedic oncologist or treating surgeon.^{17,18}

Lesion location is important for limiting the differential diagnosis. MR imaging with its excellent soft tissue contrast is superior for determining lesion location. Descriptions of lesion location include intramuscular, intermuscular, subcutaneous, and intra-articular/periarticular. A multifocal or an extensive lesion also limits diagnostic considerations to include angiomatous lesions, neurofibromatosis (NF), fibromatosis, lipomatosis, and myxoma (in cases of Mazabraud syndrome). In contradistinction to other organ locations, metastases and lymphoma are less likely considerations. Specific anatomic location may also aid in diagnosis, such as elastofibroma occurring deep to the scapular tip.

Lesions discussed in this review are included because of their frequency, location, or unique imaging characteristics, allowing a specific diagnosis or limited differential diagnosis. For common but nonspecific lesions, a reasonable differential diagnosis requires knowledge of lesion prevalence, anatomic distribution, and age range.

Lesions that predominantly affect pediatric patients (see the article by Navarro and colleagues elsewhere in this issue for further exploration of this topic), malignant soft tissue tumors (see the article by Walker and colleagues elsewhere in this issue for further exploration of this topic), and tumorlike conditions (see the article by Stacy and colleagues elsewhere in this issue for further exploration of this topic) are discussed in separate articles within this issue.

NODULAR FASCIITIS

Nodular fasciitis (**Fig. 1**) is a benign soft tissue lesion composed of proliferating fibroblasts. The lesion may grow rapidly and show high mitotic activity, simulating a more aggressive lesion. It is the most common tumor or tumorlike condition of fibrous tissue.¹⁹ Nodular fasciitis typically affects patients aged between 20 and 40 years, with no sex predilection.¹⁹⁻²² Lesions typically present as a rapidly growing painless mass that may cause mild pain or tenderness in approximately 50% of cases.¹⁹ The upper extremity is involved in 46% of cases, particularly the volar forearm. Other common locations include the head/neck (20%), the trunk (18%) and the lower extremity (16%).²³ The size of this lesion can vary from 0.5 to 10 cm, but most (71%) are 2 cm or smaller.²⁴ Nodular fasciitis has 3 common locations: subcutaneous, fascial, and intramuscular.²² Lesions are subcutaneous between 3 and 10 times more frequently than other sites. The fascial form is the second most common, and the least frequent is the intramuscular type. The deeper intramuscular form is usually larger and is the most likely to be mistaken for sarcoma.^{22,25,26} Recurrence of nodular fasciitis is rare even after partial resection.²⁴

Calcification or ossification is rarely seen on radiograph.²⁷ On T1W images, nodular fasciitis has a signal intensity similar to or slightly higher than skeletal muscle.^{25,28} With T2W sequences, the condition most often has a high signal intensity (> subcutaneous fat) but may demonstrate intermediate signal intensity.²⁶ Lesions are frequently homogeneous on T1W sequences and heterogeneous on longer repetition time (TR) acquisitions.²⁸ This lesion, as well as ancient schwannoma, is one of the few benign lesions that may demonstrate central necrosis, which may contribute to lesion heterogeneity.²³ Contrast enhancement was present in all cases in a series of 8 patients with a diffuse enhancement pattern in 63% of cases and peripheral enhancement in approximately 25%.²⁶ Linear extension along the fascia (fascial tail sign) may suggest the diagnosis, and mild surrounding edema may also be present.²³

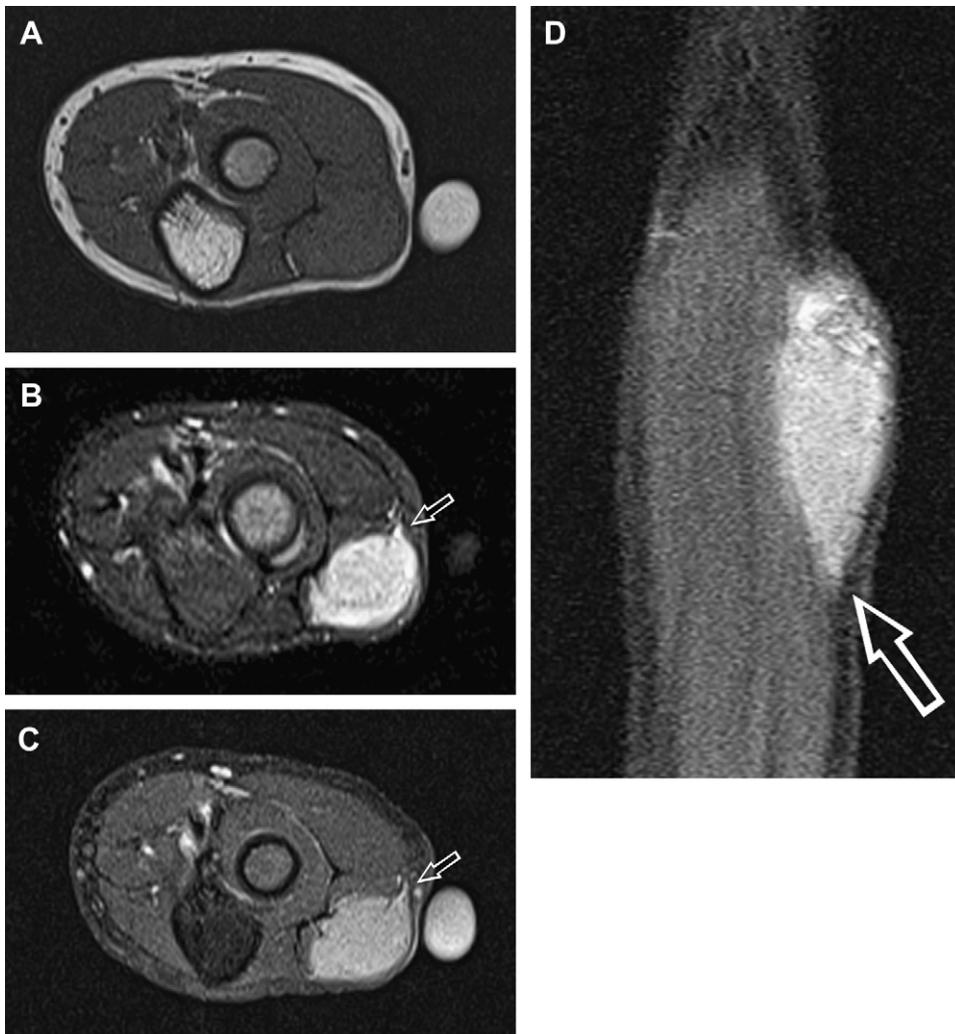


Fig. 1. Nodular fasciitis. Nodular fasciitis in a 5-year-old boy who presented with a palpable elbow mass. (A) Axial T1W (repetition time/echo time [TR/TE], 501/15) MR image demonstrates a subcutaneous soft tissue mass, which is isointense to mildly hyperintense compared with muscle. This mass is hyperintense on (B) axial short tau inversion recovery (STIR) image (TR/TE, 4700/35). Axial (C) and sagittal (D) T1W, postcontrast, fat-suppressed (TR/TE, 704/15) MR images reveal relatively homogenous diffuse enhancement. Mild linear fascial extension (fascial tail sign, *white arrows*) is demonstrated on STIR and postcontrast imaging.

The differential diagnosis on MR imaging includes benign fibrous histiocytoma, extra-abdominal desmoid tumor, neurofibroma, and malignant fibrous histiocytoma (MFH) or fibrosarcoma.

SUPERFICIAL FIBROMATOSIS: PALMAR AND PLANTAR FIBROMA

Palmar fibromatosis (Dupuytren disease) (**Fig. 2A, B**) is the most common of the superficial fibromatoses, affecting 1% to 2% of the population.²³ These lesions occur 3 to 4 times more commonly in men and most frequently in patients older than 65 years (up to 20%).^{29,30} Bilateral lesions are

present in 40% to 60% of cases.²³ The lesions are painless slow-growing palmar nodules, which may cause a flexion contracture most commonly affecting the flexor tendons of the fourth finger.³¹ Patients with palmar fibromatosis commonly have other types of fibromatoses, including plantar fibromatosis (5%–20%), Peyronie disease (2%–4%), and knuckle pad fibromatosis.^{23,30}

MR imaging typically shows multiple nodular or cordlike superficial soft tissue masses, which involve the aponeurosis of the volar aspect of the hand, extending superficially in parallel to the flexor tendons. Nodules may progress slowly (months to years) into fibrous cords, which attach

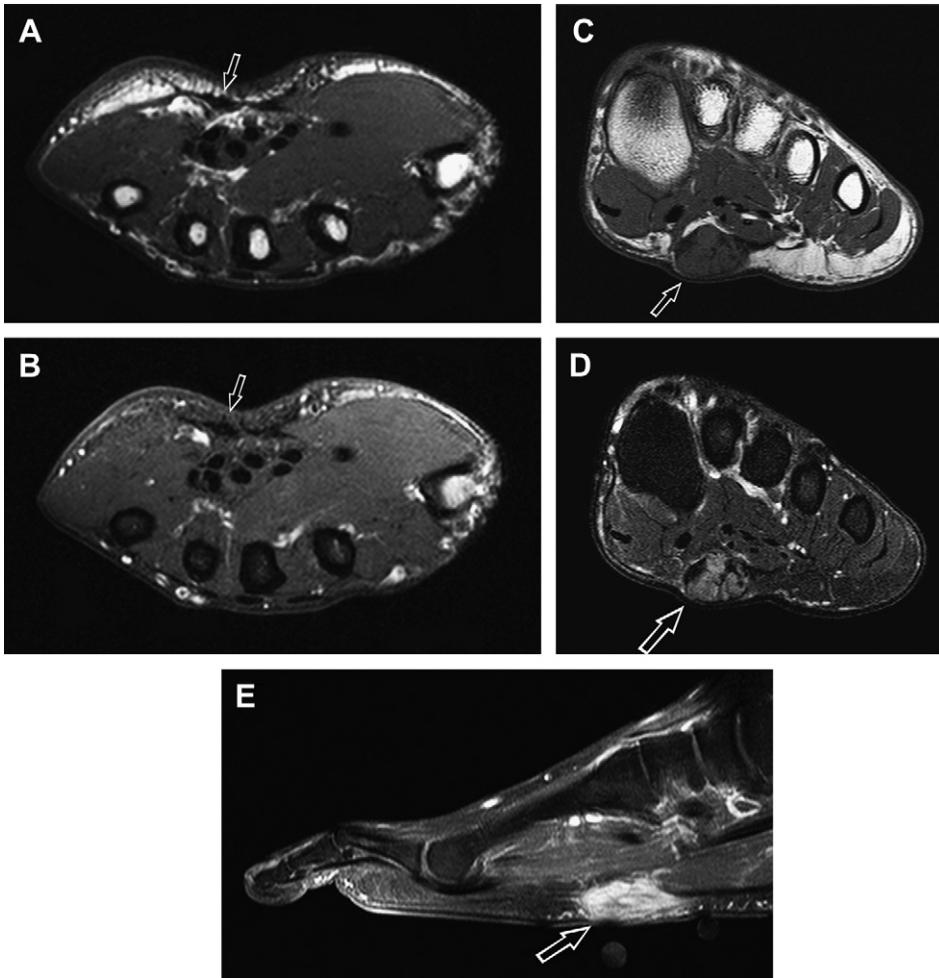


Fig. 2. Superficial fibromatosis. Palmar fibromatosis in a 44-year-old man with palmar pain at the midhypothenar eminence (A, B). (A) Axial T1W and (B) axial fat-suppressed T1W postcontrast (TR/echo time [TR/TE], 500/21) images reveal low-signal nodular thickening of the palmar fascia (arrows). (C–E) Plantar fibromatosis in a 54-year-old man with left foot pain for 1 year. MR images demonstrate a well-defined mass (arrows) in the medial aspect of the plantar aponeurosis (C). Short-axis T1W (TR/TE, 568.5/15) sequence reveals the mass with lesion signal intensity similar to skeletal muscle. There is heterogeneity with several foci of low signal within the lesion. The signal intensity of the mass is intermediate to hyperintense relative to skeletal muscle and heterogeneous on (D) short-axis T2W image with fat saturation (TR/TE, 2693.7/60), and there is marked heterogeneous enhancement on (E) sagittal fat-suppressed T1W postcontrast (TR/TE, 539.5/15) image with linear extension along the plantar aponeurosis.

to and cause traction on the underlying flexor tendons, resulting in flexion contractures of the digits (Dupuytren contractures).³² The lesion size is reported to range from 10 to 55 mm. Lesion signal intensity on T1W and T2W images is low (similar to tendon), reflecting hypocellularity and dense collagen. MR imaging can be helpful for surgical planning because relatively immature lesions demonstrate intermediate to higher signal on T1W and T2W images, reflecting the high cellularity, and have a higher local recurrence rate after local resection. Mature lesions with low T1W and T2W signal intensity are less likely to locally

recur.^{31,33,34} Lesions show diffuse enhancement, which is more prominent in lesions with higher cellularity.

Plantar fibromatosis (Ledderhose disease) (see Fig. 2C–E) occurs less frequently than the palmar lesion, with an incidence of 0.23%.³⁵ In our experience, Ledderhose disease is more frequently imaged than Dupuytren disease. Similar to palmar fibromatosis, incidence increases with advancing age, but 44% of patients were younger than 30 years in a large Armed Forces Institute of Pathology study (501 patients).^{30,36} Men are affected twice as often as women, and lesions are

bilateral in 20% to 50% of cases.^{37,38} Patients present with one or more subcutaneous nodules, which most frequently affect the medial aspect of the plantar arch (78%)³⁹ and can extend to the skin or deep structures of the foot. Nodules may be multiple in 33% of cases.³⁹ The lesions are typically painless, but patients may have pain with prolonged standing or walking.

With MR imaging, well- or ill-defined superficial lesions along the deep plantar aponeurosis typically blend with the adjacent plantar musculature. Lesions typically show heterogeneous signal (92%), which is isointense to hypointense to skeletal muscle on T1W (100%) and T2W (78%) sequences. The degree of enhancement has been reported as marked in approximately 60% and mild in 33% of cases.³⁹ Linear tails of extension (fascial tail sign) along the aponeurosis are frequent and best seen after intravenous contrast administration.^{23,32}

DEEP FIBROMATOSIS

The World Health Organization in April 2002 designated the term desmoid-type fibromatosis for all the deep fibromatoses. Desmoid tumor is a descriptive term from the Greek word *desmos* (meaning band or tendon). The biological behavior of fibromatosis is intermediate between fibroma and fibrosarcoma, although they do not

metastasize.²³ Deep or musculoaponeurotic fibromatoses include extra-abdominal fibromatosis (aggressive fibromatosis, desmoid tumor, musculoaponeurotic fibromatosis) (**Fig. 3**), abdominal fibromatosis, and intra-abdominal fibromatosis. Intra-abdominal fibromatosis arises within the pelvis and mesentery and is the type most commonly associated with Gardner syndrome.^{40,41} Abdominal fibromatosis tends to occur in women during or immediately after pregnancy or with oral contraceptive use. Estrogen seems to be a stimulatory growth factor.⁴² The rectus abdominis and internal oblique muscles of the anterior abdominal wall are most frequently affected.³⁰ The most common locations of extra-abdominal fibromatosis are the shoulder/upper arm (28%), chest wall/paraspinal region (17%), thigh (12%), and head and neck (10%–23%). These fibromatoses are most common in the second and third decades, with a peak incidence in the ages between 25 and 40 years.^{23,43} Around 2 to 4 people per million are affected with this lesion, and less than 5% are seen in the pediatric age group.³⁰ There is a female predilection in younger patients, which equalizes in older patients. Desmoid-type fibromatosis presents as a deep, firm, and poorly circumscribed soft tissue mass, which is usually slow growing and painless.^{23,30} Lesions may be multicentric in 10% to 15% of cases and may insinuate about vital neurovascular structures.^{23,44} A skeletal dysplasia has been reported

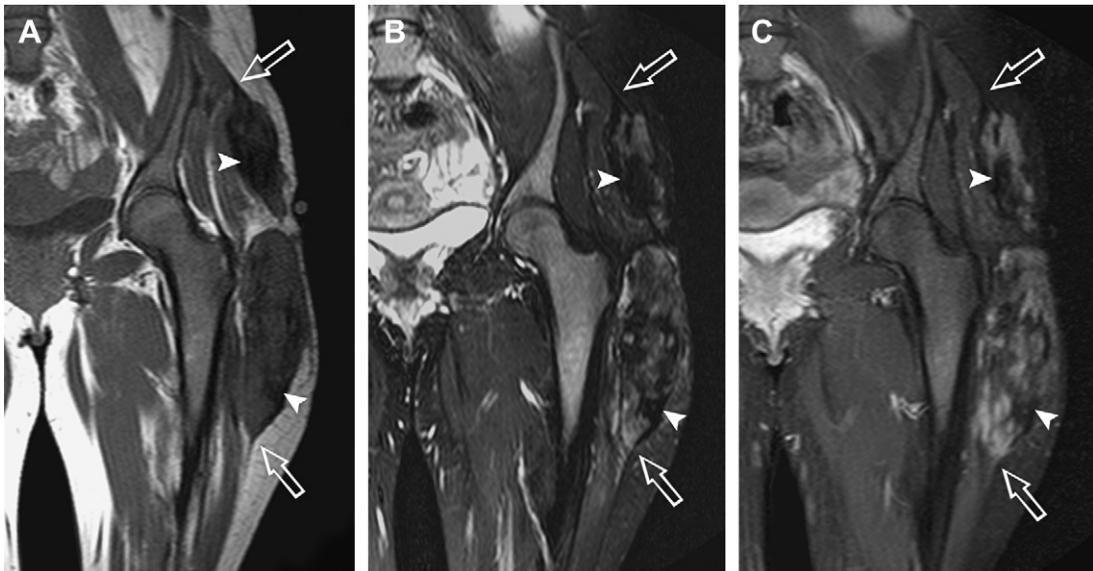


Fig. 3. Deep fibromatosis. Deep fibromatosis in a 31-year-old woman with a slowly enlarging thigh mass. (A) Coronal T1W (TR/echo time [TR/TE], 500/15), (B) T2W fat-suppressed (TR/TE, 500/15), and (C) T1W postcontrast fat-suppressed (TR/TE, 4800/70) MR images demonstrate a bilobed heterogeneous thigh mass with intermediate T1 and intermediate T2 signal, with diffuse patchy enhancement. Fascial linear extension can be seen at the proximal and distal aspect (arrows). Areas of bandlike nonenhancing low T1 and T2 signal correspond with hypocellular areas of collagen (arrowheads).

in 19% of patients with multicentric desmoid-type fibromatosis.

MR imaging is the optimal modality for evaluation of deep fibromatosis because of its superior soft tissue contrast. Lesions are usually centered intermuscularly with a rim of fat (split fat sign). Invasion of the surrounding muscle is frequent. Lesion borders are equally distributed between well-defined (49%–54%) or irregular infiltrative margins (45%–51%).^{21,32,45} Linear extension along fascial planes (fascial tail sign) is a common manifestation (80%–83% of cases).^{21,32} The signal intensity of desmoid-type fibromatosis is quite variable, reflecting the relative amounts of collagen and degree of cellularity of the lesion. Immature lesions with marked cellularity reveal higher signal intensity on long TR images. In our experience, these immature lesions are also associated with a higher local recurrence rate after resection. Relatively mature hypocellular areas with abundant collagen reveal lower signal intensity on T1W and T2W sequences often in a bandlike morphology.^{21,46} Large studies of patients have shown that the most common appearance of desmoid-type fibromatosis on MR imaging is intermediate signal intensity on both T1W (similar to muscle, 83%–95% of cases) and T2W images (lower than fat but higher than muscle on images without fat suppression, 46%–77% of cases).^{21,45,47–49} T1W and T2W sequences commonly show significant heterogeneity. Postcontrast MR imaging reveals moderate to marked heterogeneous enhancement, with less than 10% of lesions lacking significant enhancement.⁵⁰ Although low-signal T2W areas are not specific for desmoid-type fibromatosis (see suggested differential diagnosis later), the bandlike morphology of some areas of low signal intensity suggests this diagnosis, seen in 62% to 91% of cases.^{21,45} These low-signal bands are best observed on T2W or T1W fat-saturated images after intravenous gadolinium administration (the hypocellular collagenized bands do not enhance).

The differential diagnosis for soft tissue lesions with prominent areas of low signal intensity on T1W and T2W sequences includes desmoid-type fibromatosis, densely calcified masses, pigmented villonodular synovitis (PVNS)/giant cell tumor of the tendon sheath (GCTTS), elastofibroma, granular cell tumor, desmoplastic fibroblastoma, and MFH/fibrosarcoma.

ELASTOFIBROMA

Elastofibroma (**Fig. 4**) is not a neoplasm but rather a slowly growing fibroelastic reactive pseudotumor, likely resulting from mechanical friction between

the scapula and the chest wall.^{51,52} These lesions were noted in 24% of women and 11% of men in an autopsy series of patients older than 55 years.⁵³ A review of 258 chest computed tomographic (CT) examinations revealed an incidence of 2% of elastofibroma.⁵⁴ Most patients are older adults with peak incidence in the sixth and seventh decades. Lesions may be bilateral in 10% to 66% of cases.²³ There is a 2:1 female predominance. Most patients are asymptomatic (>50%), but the most common symptom is stiffness, present in 25% of cases.⁵⁵ The lesion is found between the inferior scapula tip and the chest wall in 95% to 99% of cases.⁵⁵ T1W and T2W images show a crescentic heterogeneous lesion with signal similar to adjacent skeletal muscle and streaks of tissue often at the periphery isointense to fat. T2W hypointensity is likely related to low cellularity of the fibrous tissue and elastic fibers. Heterogeneous enhancement is common.^{23,56} A key imaging feature is entrapped fat within the lesion, which is well seen with CT or MR imaging. The characteristic lesion location along with demonstration of entrapped fat is pathognomonic of elastofibroma.

LIPOMA

A lipoma is a benign neoplasm composed of mature adipose tissue. It is the most common soft tissue neoplasm and represents about 50% of all soft tissue tumors. The incidence is approximately 2.1 per 100 people.^{15,57} Lipoma is more common than liposarcoma by a ratio of 100:1.^{15,58,59} Most lipomas are discrete masses categorized by anatomic location as superficial (subcutaneous) or deep. Deep lesions are much less common and account for approximately 1% of lipomas but are imaged more frequently.¹⁵ Lipoma is rare in the first 2 decades of life.⁶⁰

Superficial lipomas (**Fig. 5A**) typically present in the fifth to seventh decades, with 80% of lesions in patients aged 27 to 85 years.⁶¹ Both men and women have been reported as more commonly affected, but there is no clear-cut sex predilection.^{59,62,63} Lesions are typically small, with 80% measuring less than 5 cm.⁵⁹ Superficial lipomas are most commonly located in the trunk, shoulders, upper arm, and neck and are unusual in the hand or foot.⁵⁹ The local recurrence rate is approximately 4%.⁶³ Superficial lipoma is often difficult to distinguish from the surrounding subcutaneous tissue, particularly if the lesion is nonencapsulated. For this reason, we prefer placing a fiducial marker over superficial lesions, position the patient so the lesion is not compressed, and

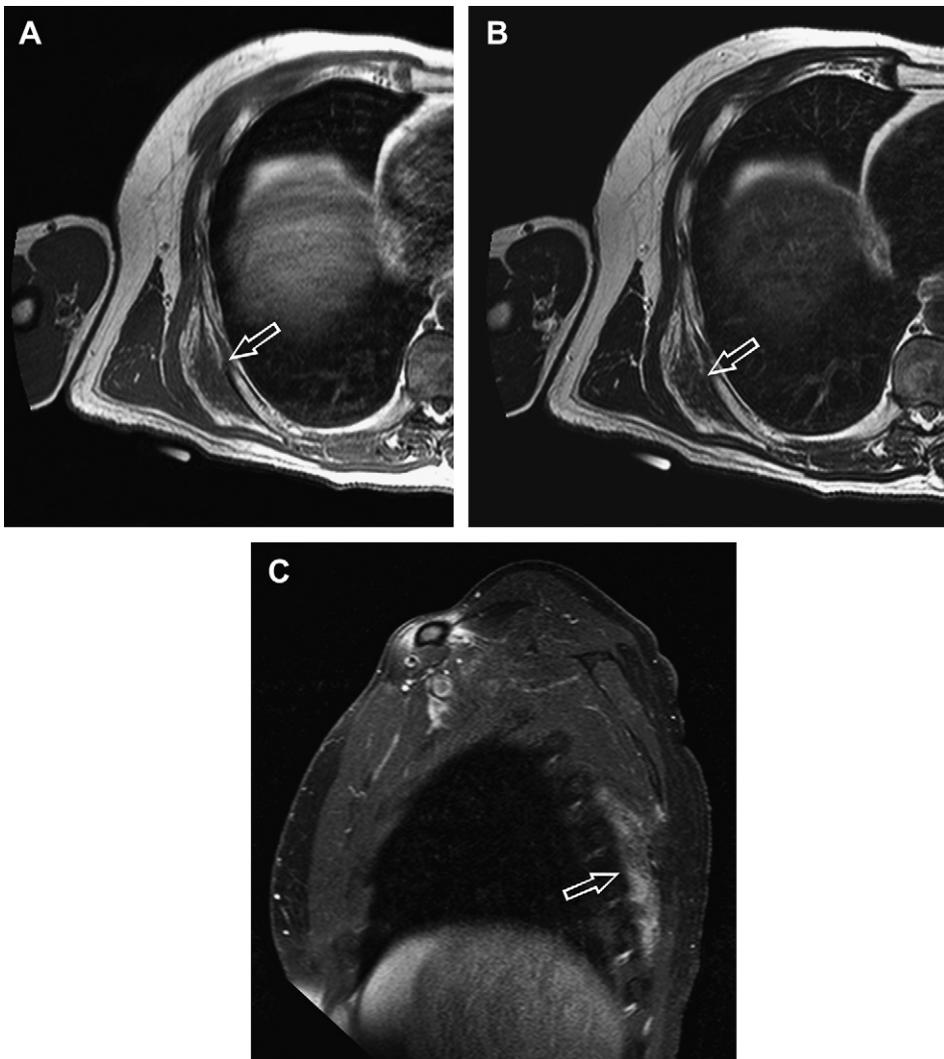


Fig. 4. Elastofibroma. Elastofibroma in a 61-year-old man with slowly growing mass beneath the right scapular tip. (A) Axial T1W (TR/echo time [TR/TE], 500/20) and (B) T2W (TR/TE, 3500/96) images without fat suppression demonstrate a lenticular mass (*white arrow*) with signal similar to skeletal muscle and hyperintense peripheral linear streaks of entrapped fat. Heterogeneous enhancement (*white arrow*) is noted in (C) sagittal T1W image after gadolinium enhancement (TR/TE, 505/7).

compare the area with the contralateral unaffected side.

Deep lipomas (including the intramuscular and intermuscular lipomatous tumors) (see **Fig. 5B, C**) occur most commonly in patients aged 20 to 60 years. Men are affected more frequently than women, and lesions commonly affect the large muscles of the lower extremity (45%), trunk (17%), shoulder (12%), and upper extremity (10%).⁶¹ Lipomas of the retroperitoneum are rare, and a lipomatous lesion in this location should be treated as a liposarcoma until proven otherwise.^{58,64} The size range of lipoma is large, and

some lesions can measure up to 20 cm.^{15,58,59} Both superficial and deep lipomas often present with a painless slow-growing soft tissue mass.⁶³ Lipomas may be multiple in 5% to 15% of patients.^{15,23,59,62} Weiss and colleagues⁶⁰ separate deep lipomas from the intramuscular and intermuscular lipomatous tumors, but we group all lipomatous lesions found beneath the superficial fascia together as deep lipomas. In our experience, deep lipomas involving the extremity are most commonly intramuscular.⁶¹

Diffuse lipomatosis is overgrowth of mature adipose tissue infiltrating through the soft tissues

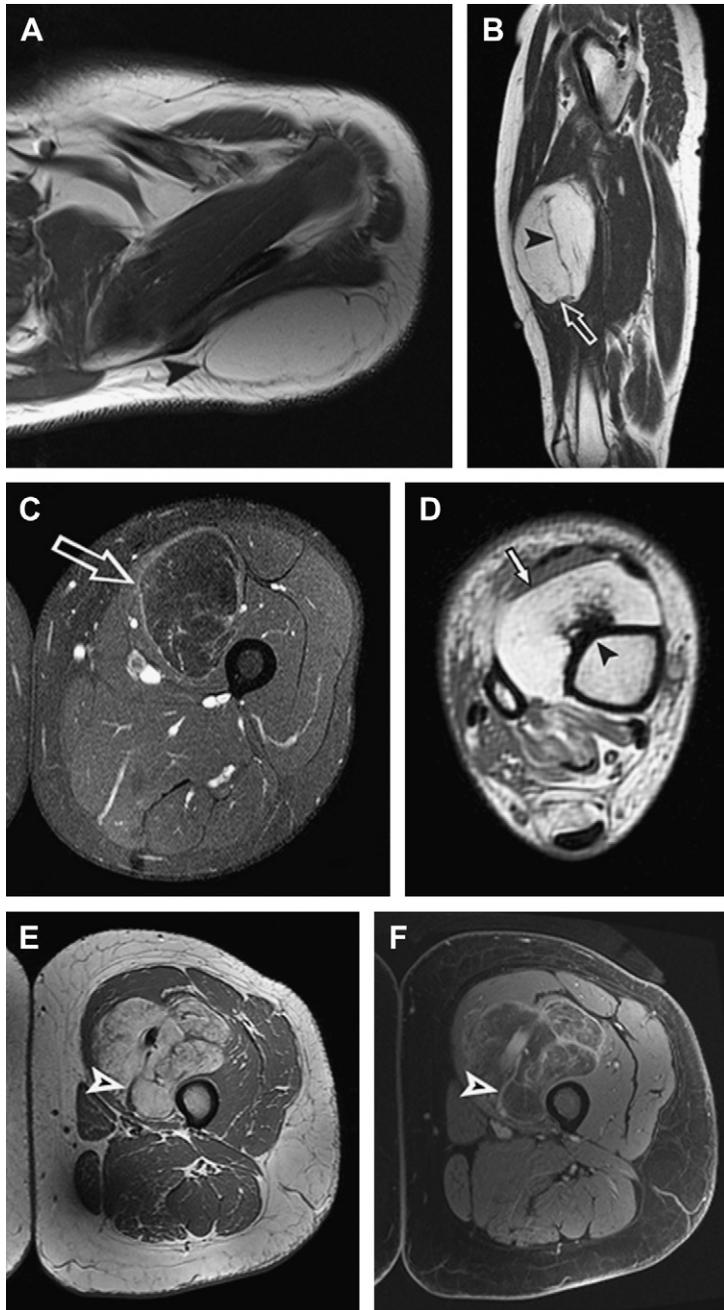


Fig. 5. Benign lipomatous lesions. Encapsulated lipoma in a 20-year-old man with enlarging shoulder mass (A). Axial T1W (TR/echo time [TR/TE], 500/11) image demonstrates a well-encapsulated (*black arrowhead*) subcutaneous lesion with signal isointense to subcutaneous fat. Intramuscular lipoma in a 38-year-old woman with slowly growing thigh mass for 6 years (B, C). (B) Sagittal T1W (TR/TE, 470.7/20) and (C) axial T1W fat-saturated postcontrast (TR/TE, 625/22) images demonstrate an intramuscular lesion (*arrows*) with signal identical to subcutaneous fat and thin fibrous septa (*black arrowhead*). Parosteal lipoma in a 41-year-old man with ankle mass (D). Axial T1W (TR/TE, 621.5/7) sequence reveals a fatty lesion (*arrow*) with high signal along the bone surface with an osseous excrescence (*arrowhead*) arising from the tibia. Note the absence of cortical and medullary continuity. Hibernoma in a 40-year-old woman with enlarging thigh mass for 5 months (E, F). (E) Axial T1W (TR/TE, 966.7/14) and (F) spoiled gradient recalled echo (TR/TE, 200/1.2) fat-saturated postcontrast sequences demonstrate an intramuscular lesion with signal intensity similar to, but not identical to, mature adult fat with prominent vessels (*arrowheads*). Unlike lipoma, serpentine vessels and prominent septa result in a lesion of greater complexity than normal subcutaneous tissue (compare with [A]).

of an affected extremity or the trunk. It is identical to lipoma microscopically.⁶⁵ Diffuse lipomatosis may be associated with osseous overgrowth and deformity.

Radiologic evaluation is diagnostic in up to 71% of cases.⁶¹ Lipomas most commonly demonstrate signal isointense to subcutaneous fat on all pulse sequences with high signal on T1W and T2W sequences and thin (<2 mm) septations; however, 28% to 30% may have thick septa or nodularity similar to liposarcoma.⁶⁶ We also find it useful to compare the degree of lesion septation to adjacent normal subcutaneous fat. Lipomas typically reveal septations of no greater thickness or number than this normal tissue. Intramuscular lipomas may have irregular margins, which interdigitate with the adjacent skeletal muscle referred to as infiltrating lipoma. In a 2003 study of 58 lipomatous lesions, lipomas showed no enhancement of septa in 58% of cases and moderate enhancement of the septa in 37%.⁶⁷ The fibrous capsule often enhances. Calcifications are reported in 11% of benign fatty tumors but are more common in malignant lesions.⁶⁸

The differential diagnosis for a lipomatous lesion with mild complexity includes lipoma, angiolipoma, myolipoma, chondroid lipoma, lipoblastoma, spindle cell/pleomorphic lipoma, hibernoma, and well-differentiated liposarcoma.

LIPOMA ARBORESCENS

Lipoma arborescens is the infiltration of subsynovial tissue by mature adipocytes. It is thought to be a reactive process frequently associated with degenerative joint disease, chronic rheumatoid arthritis, or prior trauma.⁶⁰ Clinical symptoms include painless synovial thickening and intermittent effusions.²³ Men are affected more frequently, and the age range is 9 to 66 years. The most common location is the knee.⁶⁹ The MR imaging appearance is that of a large villous frondlike mass in the suprapatellar bursa with signal similar to subcutaneous fat on all sequences and an associated joint effusion. Enhancement may be noted about these fatty fronds secondary to inflamed synovium, although typically mild.²³

HIBERNOMA

Hibernoma (see Fig. 5E, F) is a rare tumor of brown fat. These lesions usually occur between the ages of 20 and 40 years, with a peak in the third decade.⁶⁰ Hibernomas show a mild female predilection and are commonly seen in the thigh (30%), subcutaneous regions of the back (particularly the periscapular and interscapular region),

neck, axilla, shoulder, thorax, and retroperitoneum.^{70,71} The clinical presentation is usually a slow-growing painless mass that most often arises in the subcutaneous tissue, but 10% are intramuscular.⁶⁰ MR imaging features are similar to those of lipoma with prominent septations that largely represent serpentine vessels including a feeding vascular pedicle. Identification of these vascular structures by MR imaging excludes lipoma or well-differentiated liposarcoma and, in our experience, is pathognomonic of the diagnosis. Care should be taken to avoid this vascularity when these lesions are biopsied.²³ Intense uptake is reported with fludeoxyglucose F 18 positron emission tomographic scanning, which is not typically noted with lipoma or well-differentiated liposarcoma and reflects the hypervascularity and increased cellular activity of hibernoma.^{72,73}

PAROSTEAL LIPOMA

Parosteal lipoma (see Fig. 5D) represents 0.3% of all lipomas.⁷⁴ Patients are usually adults with an average age of 50 years. Parosteal lipoma shows a slight predilection for men. Lesions are usually adjacent to the diaphysis or metadiaphysis of the femur, humerus, or bones of the leg and forearm.⁷⁴ The most frequent clinical presentation is a painless nontender mass that gradually increases in size.⁷⁵ An osseous excrescence extending into a lipomatous mass or cortical thickening is noted in more than two-thirds of cases.^{23,76} MR imaging demonstrates signal identical to subcutaneous fat on all sequences. Fibrovascular septa may demonstrate high signal on long TR sequences and mild enhancement.^{75,77}

PVNS AND GCTTS

Benign proliferative lesions of the synovium, bursa, and tendon sheath represent a family of abnormalities. These lesions are believed to be benign neoplasms rather than secondary to a reactive process.^{78,79} They are divided based on their location (intra-articular vs extra-articular) and their pattern of growth (localized vs diffuse).²³ The localized or focal form of PVNS is usually extra-articular, involving the synovium about tendon sheaths or bursae. This form is often referred to as GCTTS. The diffuse form of PVNS is a monoarticular process but affects the entire synovium of a single joint.

Localized disease is approximately 7 times more common than the diffuse form of PVNS. It is typically seen in adults, in the third to fifth decades of life, with a slight female predominance.⁸⁰⁻⁸³ Localized disease most commonly

occurs in the hand and wrist (65%–89%) and clinically presents as a soft tissue swelling or a painless mobile soft tissue nodule, most frequently volar in location. It is second only to the ganglion in its frequency to cause a soft tissue mass of the hand and wrist.^{81,84} Radiographs may reveal a nonspecific soft tissue mass but are normal in 20% of patients.^{81–83} Pressure erosions of bone occur in approximately 15% of cases.^{80,83} MR imaging shows a well-defined mass intimately involving the tendon with nonspecific intrinsic signal characteristics. Lesions generally show intermediate T1W signal intensity equal to or less than muscle and T2W signal intensity equal to or

less than fat.⁸⁵ Gadolinium enhancement is noted in most cases.⁸⁶ Local recurrence after complete resection is rare.^{87–89}

The diffuse type of PVNS (**Fig. 6**) is most commonly seen in the third and fourth decades of life with an equal male and female distribution.⁹⁰ This type most commonly affects large joints, with knee involvement in 75% to 80% of patients. Less commonly, in decreasing order of frequency, the hip, ankle, shoulder, and elbow are affected.^{91–93} Involvement of more than 1 joint is rare. Patients often present with mechanical pain, swelling, and decreased range of motion because of a slow-growing mass, which worsens with activity and

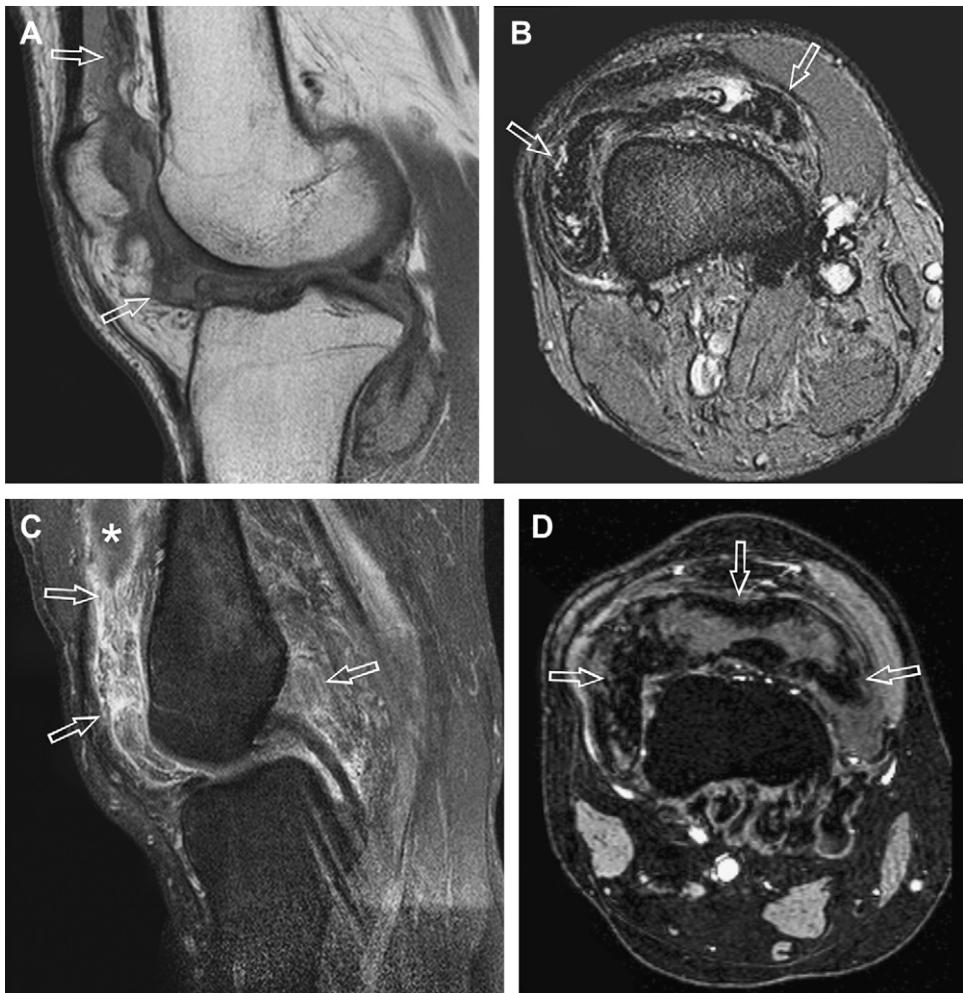


Fig. 6. PVNS. Diffuse PVNS in a 37-year-old woman with pain and swelling of right knee for 1 year. (A) Sagittal proton density-weighted (TR/echo time [TR/TE], 2000/21.3) and (B) axial fat-saturated T2W (TR/TE; 3800/105) MR images show diffuse low-signal intensity thickening (arrows) of the synovium. (C) Sagittal fat-saturated T1W postcontrast image shows diffuse synovial enhancement (arrows) throughout the knee. Note the peripheral enhancement around the joint effusion (asterisk). (D) Axial gradient echo imaging (TR/TE, 50/12) in a 26-year-old man with chronic knee pain shows accentuation (blooming) of the low signal of the thickened synovium (arrows) resulting from hemosiderin deposition.

improves with rest.⁹⁴ In contrast to the localized form, tumor recurrence after surgical resection is common, approaching 50%.⁹⁵ Pressure erosions with sclerotic borders on both sides of the joint, reflecting a synovial-based lesion, are seen in 15% of patients, more commonly in smaller less capacious joints such as the hip (93%) and shoulder (75%).^{91,92} Despite the erosions, the joint space and bone mineralization are usually preserved. On MR imaging, associated bone marrow edema may be noted at sites of osseous erosion. MR imaging characteristically shows a diffuse, heterogeneous, synovial-based thickening extending along the joint surface. On T1W imaging, the signal intensity of the mass is similar to, or slightly less than, that of skeletal muscle. Predominantly low signal intensity on T2W imaging is generally seen owing to the shortening of T2 relaxation times because of hemosiderin deposition.^{90,96,97} This low signal intensity also causes susceptibility (blooming) artifact on gradient echo imaging, helping to distinguish PVNS from other entities that may cause diffuse synovial thickening.⁹⁸ PVNS typically shows prominent enhancement after the administration of gadolinium contrast.^{99,100} Coexistent joint effusions are seen in up to 50% of cases and are usually surrounded by thickened, low-signal intensity, hemosiderin-laden synovium.¹⁰¹

BENIGN PERIPHERAL NERVE SHEATH TUMOR

Benign peripheral nerve sheath tumors (BPNST) are typically divided into schwannoma (neurilemma) and neurofibroma.⁷⁸ Both lesions contain cells closely related to the normal Schwann cell.

Schwannoma is slightly less common than neurofibroma and comprises approximately 5% of all benign soft tissue tumors.⁵⁸ Schwannoma is most commonly seen in patients aged between 20 and 50 years with an equal sex distribution.^{102,103} Schwannoma is usually a slow-growing nonaggressive lesion that presents as a painless mass smaller than 5 cm. Pain may be associated with larger lesions or schwannomatosis.^{23,102} Common sites of involvement include the cutaneous nerves of the head, neck, and flexor surface of the extremities. The posterior mediastinum and retroperitoneum are frequent locations for deep-seated lesions.¹⁰² Lesions are usually sporadic (90%) but may be plexiform or multiple in approximately 5% of cases; 3% occur with NF type 2 and 2% occur with schwannomatosis.^{104,105} The lesion is typically separable from the adjacent nerve after incising the epineurium, and nerve function is thus preserved after resection.²³

Neurofibroma (**Fig. 7A, B**) constitutes slightly more than 5% of benign soft tissue tumors.⁵⁸ Neurofibroma is most commonly seen in patients aged between 20 and 30 years and demonstrates no sex predilection.^{102,103} Three types of neurofibroma are classically described, including localized (90%), diffuse, and plexiform lesions.^{23,58} Superficial cutaneous or deep-seated nerves may be involved. Localized neurofibromas are usually slow-growing painless masses measuring less than 5 cm. The diffuse type primarily affects children and young adults and most frequently involves the subcutaneous tissue of the head and neck, and only 10% are associated with NF type 1 (NF1).^{23,58} Neurofibroma, unlike schwannoma, cannot be separated from the nerve, and complete excision of the neoplasm requires sacrifice of the nerve.¹⁰⁶

NF1 is seen in 1 in every 2500 to 3000 births.¹⁰⁷ Men are more commonly affected.¹⁰² NF1 demonstrates multiple localized neurofibromas and frequently plexiform lesions. The localized form of neurofibroma is the most common type seen with NF1. These lesions occur anywhere in the body, both superficial and deep. Plexiform neurofibromas (see **Fig. 7C**) develop (or occur) in approximately 50% of patients with NF1.¹⁰⁸ Plexiform neurofibroma represents diffuse involvement of a long segment of nerve, giving a ropelike or bag-of-worms appearance, and is pathognomonic of NF1. The incidence of malignant transformation to malignant peripheral nerve sheath tumor (MPNST) is between 2% and 29% in patients with NF1.^{102,109}

On MR imaging, the intrinsic appearance of localized lesions is nonspecific with signal intensity similar to or lower than muscle on T1W images and higher than fat on T2W images. Recognition of a well-defined fusiform shape of the lesion in a typical large nerve location can suggest that the lesion represents schwannoma, localized neurofibroma, or MPNST. The fusiform shape is caused by the tubular entering and exiting nerve.²³ With schwannoma, the entering and exiting nerve may be eccentric to the soft tissue mass. BPNST of the paraspinal region often demonstrates a dumbbell shape with extension into an enlarged neural foramina.¹¹⁰ Diffuse neurofibroma may show predominant low T2W signal, which may be related to the high collagen content. Heterogeneity of BPNST is variable, particularly with hemorrhage, necrosis, and areas of degeneration seen most commonly in the ancient schwannomas (see **Fig. 7D, E**).²³ The target sign is almost pathognomonic for neurofibroma (58%) but can be seen with schwannoma (15%). This sign refers to low to intermediate T2W signal centrally secondary to

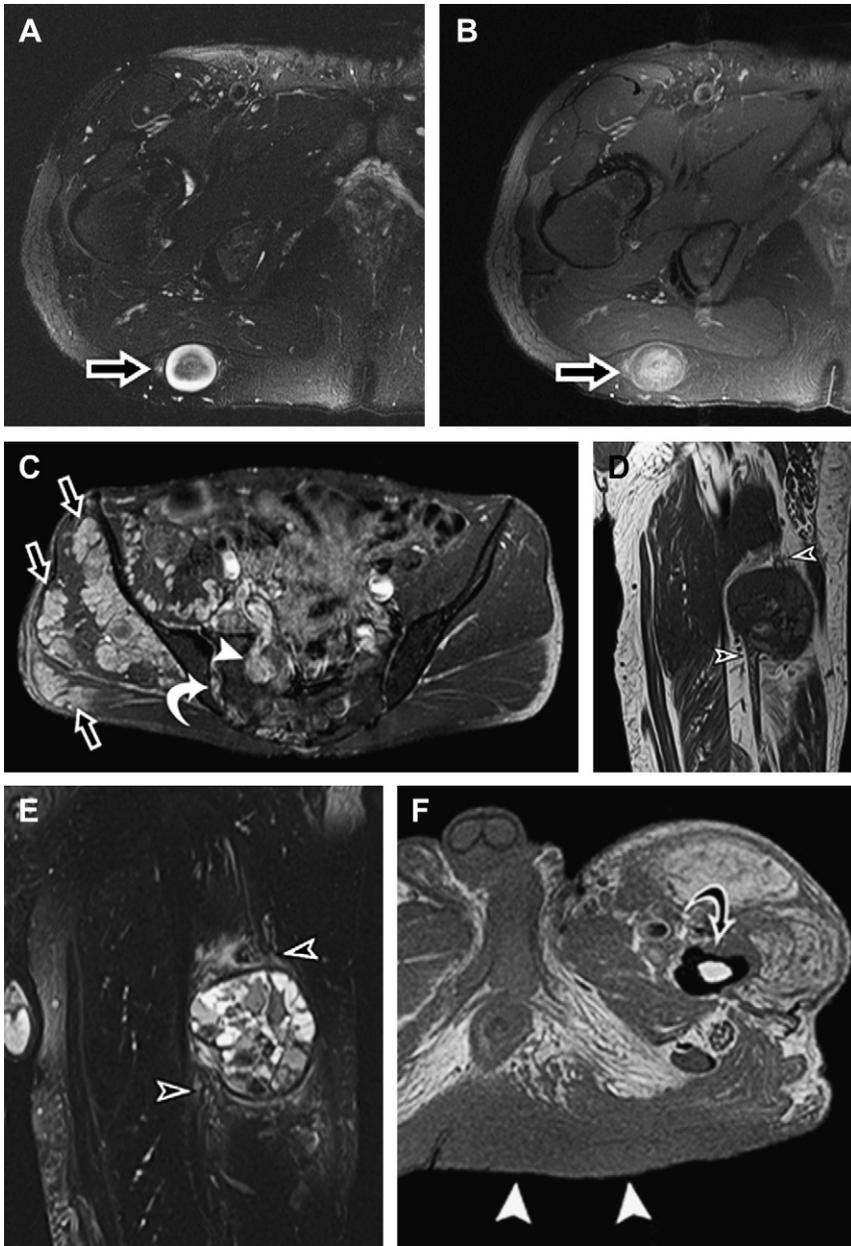


Fig. 7. Benign neural lesions. Neurofibroma in a 34-year-old man with NF1 and right buttock pain (A, B). (A) Axial T2W fat-saturated (TR/echo time [TR/TE], 4929/99) and (B) T1W fat-saturated postcontrast images (TR/TE, 750/12) demonstrate the target sign and central enhancement (arrows). The high peripheral signal on T2W images is secondary to myxoid stroma, and the fibrous center reveals marked enhancement. Plexiform neurofibromas in a 30-year-old man with NF1 (C). T1W fat-saturated postcontrast image (TR/TE; 645/14) shows plexiform neurofibromas between the gluteal muscles (arrows), in the right sacroiliac joint (curved arrow), and emerging from a right sacral foramen (arrowhead). An ancient schwannoma in a 60-year-old man with a painful left thigh mass for several months (D, E). (D) Coronal T1W (TR/TE, 560/10) and (E) T2W fat-saturated (TR/TE, 1700/25) images demonstrate a mass of the sciatic nerve with eccentric entering and exiting nerves (arrowheads). Note the marked heterogeneity from degeneration and cyst formation. Diffuse neurofibroma in a 53-year-old man with NF1 and left thigh mass for many years (F). Axial T1W image (TR/TE, 787/07) reveals replacement of the subcutaneous fat with plaque-like intermediate signal lesion (arrowheads). Also note osseous changes from mesodermal dysplasia in this patient with NF1 (curved arrow).

fibrous tissue with a higher collagen content and high T2W signal peripherally likely related to myxoid (high water content) tissue.¹¹¹ The central fibrous areas reveal contrast enhancement in neurofibroma (75%) and less frequently in schwannoma (8%).¹¹² The fascicular sign manifests as multiple ringlike structures seen on T2W or proton density-weighted images and is seen in superficial and deep-seated lesions.²³ The fascicular appearance is noted in 25% of neurofibromas and 63% of schwannomas.¹¹² A rim of fat (split fat sign) is often seen with deep-seated BPNSTs but is nonspecific and can be seen in many intermuscular lesions.¹¹³ Diffuse neurofibromas (see **Fig. 7F**) demonstrate a reticulated linear branching or plaquelike pattern within the subcutaneous tissue replacing the fat.¹¹⁴

MORTON NEUROMA

Morton neuroma (**Fig. 8**) is a nonneoplastic perineural fibrosis about the plantar digital nerve. The lesion is likely related to chronic injury. It is most commonly encountered between the third and fourth heads followed by the second and third metatarsal heads.^{102,115} The lesion exhibits a strong predilection for women (18:1) usually between the fourth and sixth decades of life and may be related to pointed and high-heeled shoe wear.^{23,116} The usual clinical presentation is paroxysmal pain often elicited by exercise and relieved by rest. The lesion is almost always

unilateral. Pain may radiate proximally or distally.^{102,116} Asymptomatic lesions are common and usually smaller than lesions causing symptoms.^{117,118}

The typical appearance is a fusiform enlargement of the plantar digital nerve plantar to the transverse metatarsal ligament, at the level of the metatarsophalangeal joint. The lesion is best imaged with MR imaging or ultrasonography. On MR imaging, Morton neuroma is best identified on the short-axis T1W sequence and is similar in signal to skeletal muscle within the intermetatarsal space on the plantar side of the transverse metatarsal ligament. T2W sequences demonstrate lesion signal less than fat, and differentiation from the surrounding muscle and fat may be difficult. Fat suppression of the fluid-sensitive sequence increases conspicuity. Enhancement of lesions is variable, occurring in 36% to 50% of lesions as reported in the literature.^{119,120} Associated intermetatarsal bursal fluid occurs in up to 67% of cases.¹²¹ Prone imaging may improve detection of Morton neuroma.¹²²

BENIGN VASCULAR LESIONS: HEMANGIOMA AND VASCULAR MALFORMATION

Hemangiomas and vascular malformations (**Fig. 9**) comprise 7% of benign soft tissue tumors and are the most common tumor in children.²³ A common classification system by Weiss and colleagues¹²³ refers to hemangioma in its broadest sense based

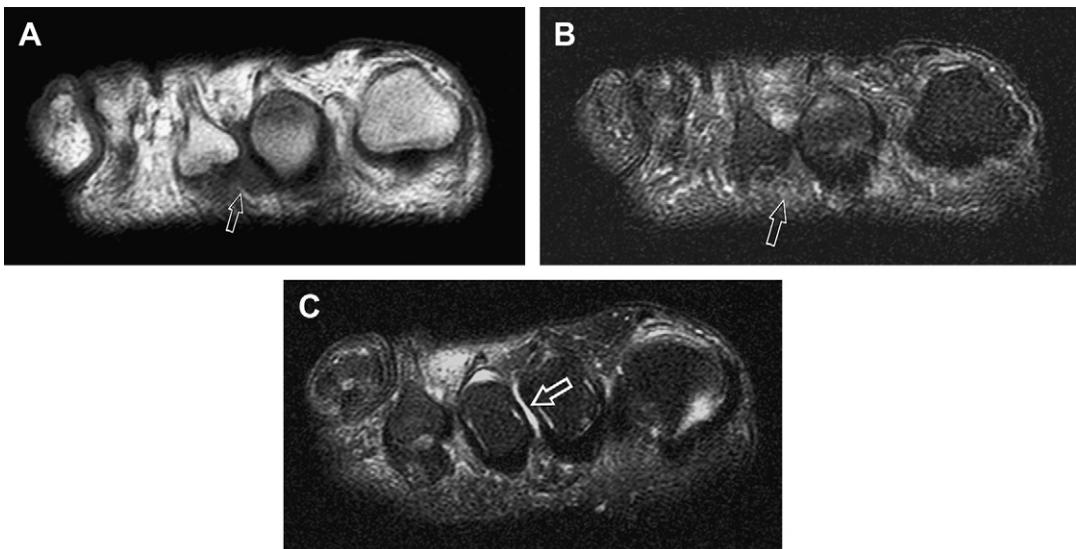


Fig. 8. Morton neuroma. Morton neuroma in a 60-year-old woman with foot pain. (A) Short-axis T1W (TR/echo time [TR/TE], 500/20) and (B) T2W (TR/TE, 2500/70) fat-suppression images demonstrate a 6-mm lesion of intermediate T1W and low T2W signal just plantar to the interspace of the second and third metatarsal heads (arrows). (C) Also note the intermetatarsal bursal fluid (arrow) revealed slightly more proximal in the forefoot on this fat-suppressed T2W image.



Fig. 9. Benign vascular malformation. Hemangioma (slow-flow vascular malformation) within the vastus medialis muscle. (A) Axial T1W image (TR/echo time [TR/TE], 500/20) and (B) sagittal T2W (TR/TE; 2500/70) image with fat suppression show heterogeneous signal intensity from fat hypertrophy (arrow [A]) and slow-flow vessels. T2W image reveals fluid levels (arrow [B]) from layering blood in slow-flow vascular channels. (C) Axial T1W (TR/TE, 570/16.7) postcontrast image shows enhancement of vascular channels (arrow).

on the common pathologic feature of a benign nonreactive lesion with an increase in the number of normal- or abnormal-appearing vessels; subdivision is based on the predominant type of vascular channel (capillary, cavernous, arteriovenous, or venous). Another classification system by Mulliken and Glowacki defines hemangiomas as true neoplasms and vascular malformations as an error in the formation of the vascular system^{124,125}; this classification system was originally based on cutaneous lesions which frequently spontaneously involute and have diagnostic clinical features and therefore are often not imaged.

Both classification systems have advantages and disadvantages regarding their use that are beyond the scope of discussion for this article. The Mulliken/Glowacki classification is useful for

clinicians and allows accurate diagnosis of neoplasm versus malformation based on history and physical examination. As it is based on lesions of infancy and childhood, it is discussed in detail in Dr Navarro's article "Soft Tissue Masses in Children" in this issue. In the typical adult patient, we find that the system proposed by Weiss and Goldblum is most applicable for benign vascular lesions occurring in deeper soft tissues such as muscles and joints. We henceforth refer to these lesions as hemangiomas, acknowledging that the distinction between hemangioma and vascular malformation is not always straightforward and that therefore some hemangiomas may represent true malformations (eg, venous malformations as described by Mulliken and Glowacki) rather than tumors.¹²⁶

Of the nonregressing vascular lesions, cavernous hemangiomas (as defined by Weiss and colleagues) are the most common. These lesions may be superficial or deep. Superficial lesions may cause bluish skin discoloration. Deep lesions are more commonly intramuscular, have nonspecific clinical features, and are usually referred for radiologic evaluation. These lesions are often considered congenital in origin and grow at the same rate as normal tissues. Occurrence is more common in women than men by a ratio of 3:1, and growth may occur during pregnancy.¹²³ Clinical presentation depends on location, and the lesion may be painful and may change size with engorgement. Intramuscular lesions may be painful with exercise, presumably because of local muscle ischemia.

These lesions are commonly imaged when symptomatic and because they do not involute. Radiographs may be normal or may show a soft tissue mass and phleboliths (20%–67% of cases).²³ Reactive and pressure changes of bone may occur, particularly when lesions are adjacent to bone, and include benign periosteal reaction (23%), cortical scalloping, and linear lucencies (31%).¹²⁷ Nonenhanced CT shows a soft tissue density mass with or without phleboliths. MR imaging features are often characteristic. The lesion may be well defined or infiltrative. Lesions

show low to intermediate signal intensity on T1W images. Associated fatty overgrowth due to chronically ischemic muscle is common in deep-seated lesions and follows subcutaneous adipose signal. Intralesional hemorrhage may occur, showing areas of high T1 signal and rarely fluid levels. Vascular elements show high signal intensity on T2W images and are typically serpentine in morphology, and thus lesions are often prominently heterogeneous. Enhancement is prominent, and feeding vessels may be evident. In our experience, approximately 90% of deep hemangiomas reveal these pathognomonic features of serpentine vascular channels and fat overgrowth and do not require biopsy for diagnosis.

Angiomatosis represents diffuse infiltration by hemangiomas and/or lymphangiomas. Imaging characteristics are similar to solitary lesions except for the distribution with involvement of multiple soft tissue planes and prominent longitudinal extension. Many angiomatous syndromes have been described. Most of these syndromes are without malignant potential with the exception of Maffucci syndrome.

GLOMUS TUMOR

Glomus tumor (**Fig. 10**) is a neoplasm that develops from the neuromyoarterial glomus

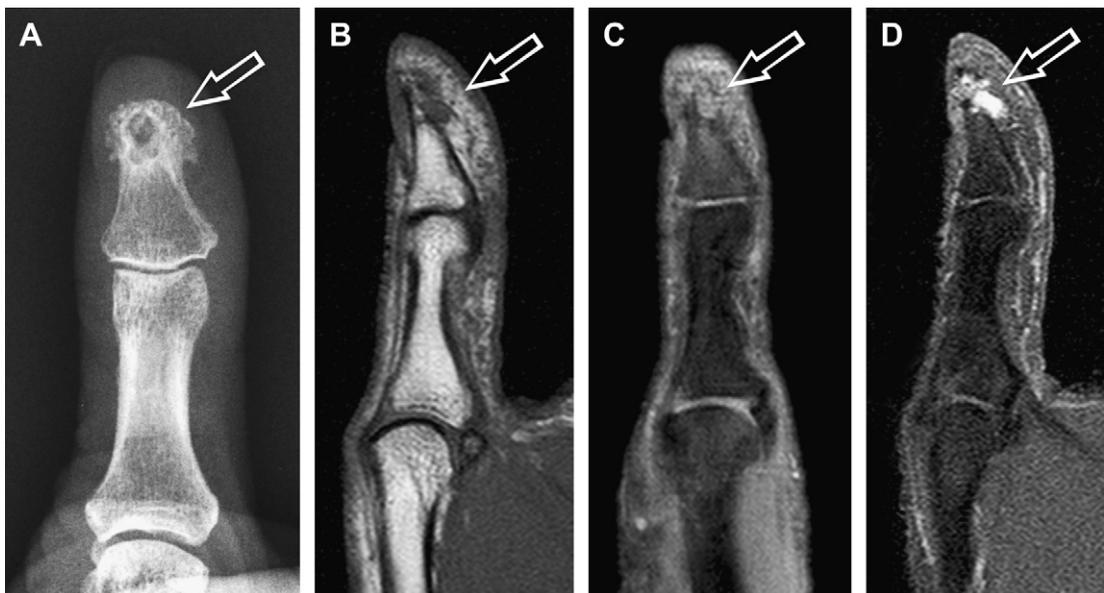


Fig. 10. Glomus tumor. Glomus tumor (arrow) in a 57-year-old woman with left thumb pain. (A) Anteroposterior radiograph of the thumb demonstrates prominent bone erosion of the terminal tuft. (B) Sagittal T1W (TR/echo time [TR/TE], 760/16.7) MR image demonstrates an intermediate-signal 6-mm mass on the volar aspect of the terminal tuft. The lesion is intermediate signal on (C) coronal proton density-weighted (TR/TE, 1500/10) fat-saturated image and demonstrates avid contrast enhancement on (D) sagittal T1W (TR/TE, 570/16.7) image with fat saturation.

body.^{23,128} The estimated incidence is 1.6% of soft tissue tumors. There is no gender predilection overall, but there is a 3:1 female predominance for subungual lesions.¹²⁹ Multiple glomus tumors (nearly 10% of patients) may be present in NF1.^{130,131} The lesion is most frequently diagnosed between 20 and 40 years of age. The most common site is the subungual location (65%)¹³² in the finger, but other locations include the palm, wrist, forearm, and foot.¹³³ The average lesion size is approximately 7 mm in the upper extremity and 13 mm in the lower extremity in a recent series.¹³² The most frequent clinical presentation is a small red-blue nodule causing paroxysms of pain radiating away from the lesion, which is often elicited by cold or pressure. The classic clinical triad of pain, point tenderness, and cold sensitivity is present in approximately 30% of cases.¹³⁴

Imaging reveals a small mass related to the nail bed, with erosion of bone in 22% to 82% of cases.²³ MR imaging reveals a small mass with homogeneous high signal on T2W^{135,136} and intermediate to low signal intensity noted on T1W images.¹²⁸ Rarely, glomus tumors may show cystic change.¹³⁷ Enhancement is typically prominent and diffuse. A high-resolution surface coil has proven useful to demonstrate cortical bone erosion.¹³⁴

MYXOMA

Myxoma is a mesenchymal neoplasm composed of undifferentiated stellate cells in a myxoid

stroma.¹³⁸ The lesion is most frequently seen in adults aged between 40 and 70 years, with a female predilection (67%).^{43,139} Patients typically present with a painless soft tissue mass. Intramuscular myxomas (Fig. 11) are usually solitary. Multiple myxomas are almost always associated with monostotic or polyostotic fibrous dysplasia (Mazabraud syndrome).¹⁴⁰ In a study of 200 myxomas from various sites, 17% were noted to be intramuscular.¹⁴¹ Most musculoskeletal myxomas are intramuscular (82%), with the thigh (51%), upper arm (9%), calf (7%), and buttock (7%) being the most frequent locations. A small number of lesions are intermuscular (9%), subcutaneous (9%), or juxta-articular (7%).¹⁴² Myxomas show low (81%–100%) to intermediate (0%–19%) signal intensity on T1W images. On T2W sequences, all myxomas demonstrate high signal intensity. Lesions are homogeneous or only mildly heterogeneous and are well defined in 60% to 80% of cases. In 65% to 89% of cases, a thin rim of fat is noted most prominent at the superior and inferior aspects of the lesion, representing atrophy of the adjacent muscle. Perilesional high signal may be noted on fluid-sensitive sequences in 79% to 100% of myxomas caused by leakage of the myxomatous tissue into the surrounding muscle causing edema. Myxomas reveal mild (76%) to moderate (24%) contrast enhancement in a diffuse (57%) or thick peripheral and septal (43%) enhancement pattern. Cystic areas may be noted in slightly more than 50% of all myxomas.^{142–145} The MR imaging appearance of an intramuscular lesion with low T1W signal

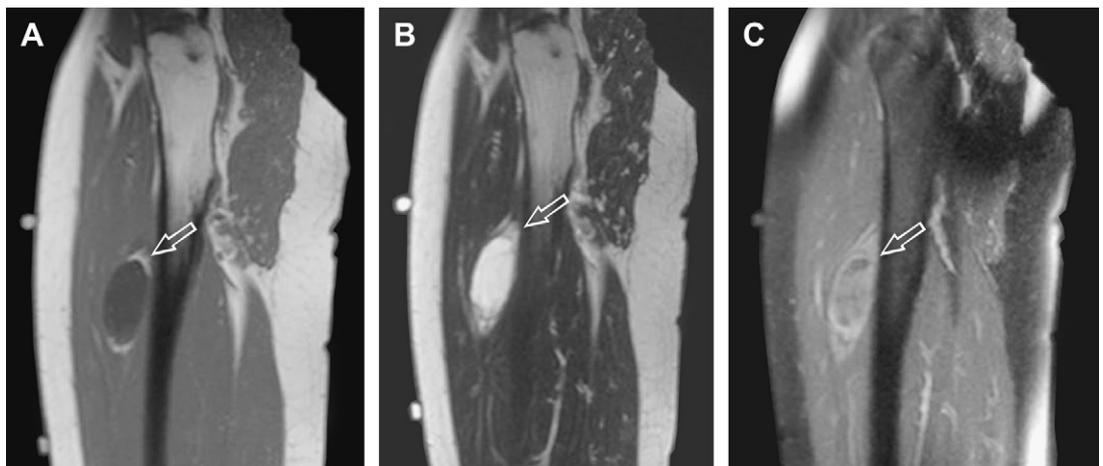


Fig. 11. Intramuscular myxoma. Intramuscular myxoma in a 49-year-old woman with palpable right anterior thigh mass. (A) Sagittal T1W (TR/echo time [TR/TE], 596/13) and (B) sagittal T2W (TR/TE, 5080/100) images demonstrate a well-defined cystlike mass with low T1 signal and high T2 signal. Note the surrounding edema on the T2 sequence most prominent at the superior and inferior poles of the lesion. Sagittal T1W (C) fat-saturated image after gadolinium enhancement (TR/TE, 500/13) reveals mild diffuse enhancement of the lesion.

and high signal intensity on fluid-sensitive sequences demonstrating a peripheral rim of fat and edema is highly suggestive if not pathognomonic for myxoma.

The differential diagnosis for a predominantly myxoid-appearing soft tissue lesion includes myxoid liposarcoma, myxofibrosarcoma (myxoid MFH), myxoid chondrosarcoma, myxoma, ganglion, synovial cyst, and peripheral nerve sheath tumor.

This has been a limited review of common benign soft tissue tumors in the adult population. In general, small-sized (<5 cm) well-defined margins and homogeneous MR imaging signal favor a benign lesion. When a specific diagnosis is not possible, a solid lesion should be considered indeterminate (even one with all the benign features mentioned earlier) and the appropriate biopsy path planned with the treating surgeon.

ACKNOWLEDGMENTS

We gratefully acknowledge the residents who attend the American Institute for Radiologic Pathology courses (past, present, and future) for their contribution of interesting cases to our series of patients, which allow our continued educational outreach.

REFERENCES

- Hajdu SI. Benign soft tissue tumors: classification and natural history. *CA Cancer J Clin* 1987;37(2):66–76.
- Walker EA, Song AJ, Murphey MD. Magnetic resonance imaging of soft-tissue masses. *Semin Roentgenol* 2010;45(4):277–97.
- Beltran J, Chandnani V, McGhee RA Jr, et al. Gadopentetate dimeglumine-enhanced MR imaging of the musculoskeletal system. *AJR Am J Roentgenol* 1991;156(3):457–66.
- Verstraete KL, De Deene Y, Roels H, et al. Benign and malignant musculoskeletal lesions: dynamic contrast-enhanced MR imaging—parametric “first-pass” images depict tissue vascularization and perfusion. *Radiology* 1994;192(3):835–43.
- Benedikt RA, Jelinek JS, Kransdorf MJ, et al. MR imaging of soft-tissue masses: role of gadopentetate dimeglumine. *J Magn Reson Imaging* 1994;4(3):485–90.
- Erlmann R, Reiser MF, Peters PE, et al. Musculoskeletal neoplasms: static and dynamic Gd-DTPA-enhanced MR imaging. *Radiology* 1989;171(3):767–73.
- van Rijswijk CS, Geirnaerd MJ, Hogendoorn PC, et al. Soft-tissue tumors: value of static and dynamic gadopentetate dimeglumine-enhanced MR imaging in prediction of malignancy. *Radiology* 2004;233(2):493–502.
- Mirowitz SA, Totty WG, Lee JK. Characterization of musculoskeletal masses using dynamic Gd-DTPA enhanced spin-echo MRI. *J Comput Assist Tomogr* 1992;16(1):120–5.
- Berquist TH, Ehman RL, King BF, et al. Value of MR imaging in differentiating benign from malignant soft-tissue masses: study of 95 lesions. *AJR Am J Roentgenol* 1990;155(6):1251–5.
- Sundaram M, McGuire MH, Herbold DR. Magnetic resonance imaging of soft tissue masses: an evaluation of fifty-three histologically proven tumors. *Magn Reson Imaging* 1988;6(3):237–48.
- Moulton JS, Blebea JS, Dunco DM, et al. MR imaging of soft-tissue masses: diagnostic efficacy and value of distinguishing between benign and malignant lesions. *AJR Am J Roentgenol* 1995;164(5):1191–9.
- Totty WG, Murphy WA, Lee JK. Soft-tissue tumors: MR imaging. *Radiology* 1986;160(1):135–41.
- Kransdorf MJ, Jelinek JS, Moser RP Jr, et al. Soft-tissue masses: diagnosis using MR imaging. *AJR Am J Roentgenol* 1989;153(3):541–7.
- Crim JR, Seeger LL, Yao L, et al. Diagnosis of soft-tissue masses with MR imaging: can benign masses be differentiated from malignant ones? *Radiology* 1992;185(2):581–6.
- Myhre-Jensen O. A consecutive 7-year series of 1331 benign soft tissue tumours. Clinicopathologic data. Comparison with sarcomas. *Acta Orthop Scand* 1981;52(3):287–93.
- Rydholm A. Management of patients with soft-tissue tumors. Strategy developed at a regional oncology center. *Acta Orthop Scand Suppl* 1983;203:13–77.
- Mankin HJ, Lange TA, Spanier SS. The hazards of biopsy in patients with malignant primary bone and soft-tissue tumors. *J Bone Joint Surg Am* 1982;64(8):1121–7.
- Mankin HJ, Mankin CJ, Simon MA. The hazards of the biopsy, revisited. Members of the Musculoskeletal Tumor Society. *J Bone Joint Surg Am* 1996;78(5):656–63.
- Weiss SW, Goldblum JR, Enzinger FM. Benign fibroblastic/myofibroblastic proliferations. In: Weiss SW, Goldblum JR, editors. *Enzinger and Weiss' soft tissue tumors*. 5th edition. Philadelphia: Mosby Elsevier; 2008. p. 175–225.
- Meister P, Buckmann FW, Konrad E. Nodular fasciitis (analysis of 100 cases and review of the literature). *Pathol Res Pract* 1978;162(2):133–65.
- Dinauer PA, Brixey CJ, Moncur JT, et al. Pathologic and MR imaging features of benign fibrous soft-tissue tumors in adults. *Radiographics* 2007;27(1):173–87.

22. Shimizu S, Hashimoto H, Enjoji M. Nodular fasciitis: an analysis of 250 patients. *Pathology* 1984;16(2):161–6.
23. Kransdorf MJ, Murphey MD. Imaging of soft tissue tumors. 2nd edition. Philadelphia: Lippincott Williams & Wilkins; 2006.
24. Bernstein KE, Lattes R. Nodular (pseudosarcomatous) fasciitis, a nonrecurrent lesion: clinicopathologic study of 134 cases. *Cancer* 1982;49(8):1668–78.
25. Leung LY, Shu SJ, Chan AC, et al. Nodular fasciitis: MRI appearance and literature review. *Skeletal Radiol* 2002;31(1):9–13.
26. Wang XL, De Schepper AM, Vanhoenacker F, et al. Nodular fasciitis: correlation of MRI findings and histopathology. *Skeletal Radiol* 2002;31(3):155–61.
27. Broder MS, Leonidas JC, Mitty HA. Pseudosarcomatous fasciitis: an unusual cause of soft-tissue calcification. *Radiology* 1973;107(1):173–4.
28. Meyer CA, Kransdorf MJ, Jelinek JS, et al. MR and CT appearance of nodular fasciitis. *J Comput Assist Tomogr* 1991;15(2):276–9.
29. Mikkelsen OA. Dupuytren's disease—initial symptoms, age of onset and spontaneous course. *Hand* 1977;9(1):11–5.
30. Weiss SW, Goldblum JR, Enzinger FM. Fibromatoses. In: Weiss SW, Goldblum JR, editors. *Enzinger and Weiss' soft tissue tumors*. 5th edition. Philadelphia: Mosby Elsevier; 2008. p. 227–8.
31. Yacoe ME, Bergman AG, Ladd AL, et al. Dupuytren's contracture: MR imaging findings and correlation between MR signal intensity and cellularity of lesions. *AJR Am J Roentgenol* 1993;160(4):813–7.
32. Murphey MD, Ruble CM, Tyszko SM, et al. From the archives of the AFIP: musculoskeletal fibromatoses: radiologic-pathologic correlation. *Radiographics* 2009;29(7):2143–73.
33. Robbin MR, Murphey MD, Temple HT, et al. Imaging of musculoskeletal fibromatosis. *Radiographics* 2001;21(3):585–600.
34. Rombouts JJ, Noel H, Legrain Y, et al. Prediction of recurrence in the treatment of Dupuytren's disease: evaluation of a histologic classification. *J Hand Surg Am* 1989;14(4):644–52.
35. Yost J, Winters T, Fett HC, et al. Dupuytren's contracture; a statistical study. *Am J Surg* 1955;90(4):568–71.
36. Fetsch JF, Laskin WB, Miettinen M. Palmar-plantar fibromatosis in children and preadolescents: a clinicopathologic study of 56 cases with newly recognized demographics and extended follow-up information. *Am J Surg Pathol* 2005;29(8):1095–105.
37. Lee TH, Wapner KL, Hecht PJ. Plantar fibromatosis. *J Bone Joint Surg Am* 1993;75(7):1080–4.
38. Aviles E, Arlen M, Miller T. Plantar fibromatosis. *Surgery* 1971;69(1):117–20.
39. Morrison WB, Schweitzer ME, Wapner KL, et al. Plantar fibromatosis: a benign aggressive neoplasm with a characteristic appearance on MR images. *Radiology* 1994;193(3):841–5.
40. Clark SK, Phillips RK. Desmoids in familial adenomatous polyposis. *Br J Surg* 1996;83(11):1494–504.
41. Jones IT, Jagelman DG, Fazio VW, et al. Desmoid tumors in familial polyposis coli. *Ann Surg* 1986;204(1):94–7.
42. Pritchard DJ, Nascimento AG, Petersen IA. Local control of extra-abdominal desmoid tumors. *J Bone Joint Surg Am* 1996;78(6):848–54.
43. Fletcher CDM, Unni KK, Mertens F. World Health Organization pathology and genetics of tumors of soft tissue and bone. 3rd edition. Lyon (France): IARC Press; 2006.
44. Rock MG, Pritchard DJ, Reiman HM, et al. Extra-abdominal desmoid tumors. *J Bone Joint Surg Am* 1984;66(9):1369–74.
45. Lee JC, Thomas JM, Phillips S, et al. Aggressive fibromatosis: MRI features with pathologic correlation. *AJR Am J Roentgenol* 2006;186(1):247–54.
46. Sundaram M, McGuire MH, Schajowicz F. Soft-tissue masses: histologic basis for decreased signal (short T2) on T2-weighted MR images. *AJR Am J Roentgenol* 1987;148(6):1247–50.
47. Kransdorf MJ, Jelinek JS, Moser RP Jr, et al. Magnetic resonance appearance of fibromatosis. A report of 14 cases and review of the literature. *Skeletal Radiol* 1990;19(7):495–9.
48. Feld R, Burk DL Jr, McCue P, et al. MRI of aggressive fibromatosis: frequent appearance of high signal intensity on T2-weighted images. *Magn Reson Imaging* 1990;8(5):583–8.
49. Quinn SF, Erickson SJ, Dee PM, et al. MR imaging in fibromatosis: results in 26 patients with pathologic correlation. *AJR Am J Roentgenol* 1991;156(3):539–42.
50. Romero JA, Kim EE, Kim CG, et al. Different biologic features of desmoid tumors in adult and juvenile patients: MR demonstration. *J Comput Assist Tomogr* 1995;19(5):782–7.
51. Jarvi O, Saxen E. Elastofibroma dorsi. *Acta Pathol Microbiol Scand Suppl* 1961;51(Suppl 144):83–4.
52. Jarvi OH, Saxen AE, Hopsu-Havu VK, et al. Elastofibroma—a degenerative pseudotumor. *Cancer* 1969;23(1):42–63.
53. Jarvi OH, Lansimies PH. Subclinical elastofibromas in the scapular region in an autopsy series. *Acta Pathol Microbiol Scand A* 1975;83(1):87–108.
54. Brandser EA, Goree JC, El-Khoury GY. Elastofibroma dorsi: prevalence in an elderly patient population as revealed by CT. *AJR Am J Roentgenol* 1998;171(4):977–80.
55. Nagamine N, Nohara Y, Ito E. Elastofibroma in Okinawa. A clinicopathologic study of 170 cases. *Cancer* 1982;50(9):1794–805.
56. Faccioli N, Foti G, Comai A, et al. MR imaging findings of elastofibroma dorsi in correlation with pathological features: our experience. *Radiol Med* 2009;114(8):1283–91.

57. Ronan SJ, Broderick T. Minimally invasive approach to familial multiple lipomatosis. *Plast Reconstr Surg* 2000;106(4):878–80.
58. Kransdorf MJ. Benign soft-tissue tumors in a large referral population: distribution of specific diagnoses by age, sex, and location. *AJR Am J Roentgenol* 1995;164(2):395–402.
59. Rydholm A, Berg NO. Size, site and clinical incidence of lipoma. Factors in the differential diagnosis of lipoma and sarcoma. *Acta Orthop Scand* 1983;54(6):929–34.
60. Weiss SW, Goldblum JR, Enzinger FM. Benign lipomatous tumors. In: Weiss SW, Goldblum JR, editors. *Enzinger and Weiss' soft tissue tumors*. 5th edition. Philadelphia: Mosby Elsevier; 2008. p. 429–76.
61. Murphey MD, Carroll JF, Flemming DJ, et al. From the archives of the AFIP: benign musculoskeletal lipomatous lesions. *Radiographics* 2004;24(5):1433–66.
62. Kransdorf MJ, Moser RP Jr, Meis JM, et al. Fat-containing soft-tissue masses of the extremities. *Radiographics* 1991;11(1):81–106.
63. Leffert RD. Lipomas of the upper extremity. *J Bone Joint Surg Am* 1972;54(6):1262–6.
64. Kransdorf MJ. Malignant soft-tissue tumors in a large referral population: distribution of diagnoses by age, sex, and location. *AJR Am J Roentgenol* 1995;164(1):129–34.
65. Ha TV, Kleinman PK, Fraire A, et al. MR imaging of benign fatty tumors in children: report of four cases and review of the literature. *Skeletal Radiol* 1994;23(5):361–7.
66. Murphey MD, Arcara LK, Fanburg-Smith J. From the archives of the AFIP: imaging of musculoskeletal liposarcoma with radiologic-pathologic correlation. *Radiographics* 2005;25(5):1371–95.
67. Ohguri T, Aoki T, Hisaoka M, et al. Differential diagnosis of benign peripheral lipoma from well-differentiated liposarcoma on MR imaging: is comparison of margins and internal characteristics useful? *AJR Am J Roentgenol* 2003;180(6):1689–94.
68. Kransdorf MJ, Bancroft LW, Peterson JJ, et al. Imaging of fatty tumors: distinction of lipoma and well-differentiated liposarcoma. *Radiology* 2002;224(1):99–104.
69. Armstrong SJ, Watt I. Lipoma arborescens of the knee. *Br J Radiol* 1989;62(734):178–80.
70. Evers LH, Gebhard M, Lange T, et al. Hibernoma—case report and literature review. *Am J Dermatopathol* 2009;31(7):685–6.
71. Furlong MA, Fanburg-Smith JC, Miettinen M. The morphologic spectrum of hibernoma: a clinicopathologic study of 170 cases. *Am J Surg Pathol* 2001;25(6):809–14.
72. Nishida J, Ehara S, Shiraishi H, et al. Clinical findings of hibernoma of the buttock and thigh: rare involvements and extremely high uptake of FDG-PET. *Med Sci Monit* 2009;15(7):CS117–22.
73. Smith CS, Teruya-Feldstein J, Caravelli JF, et al. False-positive findings on 18F-FDG PET/CT: differentiation of hibernoma and malignant fatty tumor on the basis of fluctuating standardized uptake values. *AJR Am J Roentgenol* 2008;190(4):1091–6.
74. Goldman AB, DiCarlo EF, Marcove RC. Case report 774. Coincidental parosteal lipoma with osseous excrescence and intramuscular lipoma. *Skeletal Radiol* 1993;22(2):138–45.
75. Murphey MD, Johnson DL, Bhatia PS, et al. Parosteal lipoma: MR imaging characteristics. *AJR Am J Roentgenol* 1994;162(1):105–10.
76. Bui-Mansfield LT, Myers CP, Chew FS. Parosteal lipoma of the fibula. *AJR Am J Roentgenol* 2000;174(6):1698.
77. Yu JS, Weis L, Becker W. MR imaging of a parosteal lipoma. *Clin Imaging* 2000;24(1):15–8.
78. Weiss SW, Goldblum JR, Enzinger FM. Benign tumors and tumor-like lesions of synovial tissue. In: *Enzinger and Weiss's soft tissue tumors*. 4th edition. St Louis (MO): Mosby; 2001. p. 1037–62.
79. Jaffe HL, Lichtenstein L, Sutro CJ. Pigmented villonodular synovitis, bursitis and tenosynovitis. *Arch Pathol Lab Med* 1941;31:731–65.
80. Ushijima M, Hashimoto H, Tsuneyoshi M, et al. Giant cell tumor of the tendon sheath (nodular tenosynovitis). A study of 207 cases to compare the large joint group with the common digit group. *Cancer* 1986;57(4):875–84.
81. Savage RC, Mustafa EB. Giant cell tumor of tendon sheath (localized nodular tenosynovitis). *Ann Plast Surg* 1984;13(3):205–10.
82. Oyemade GA, Abioye AA. A clinicopathologic review of benign giant cell tumors of tendon sheaths in Ibadan, Nigeria. *Am J Surg* 1977;134(3):392–5.
83. Karasick D, Karasick S. Giant cell tumor of tendon sheath: spectrum of radiologic findings. *Skeletal Radiol* 1992;21(4):219–24.
84. Bogumill GP, Sullivan DJ, Baker GI. Tumors of the hand. *Clin Orthop Relat Res* 1975;(108):214–22.
85. Jelinek JS, Kransdorf MJ, Shmookler BM, et al. Giant cell tumor of the tendon sheath: MR findings in nine cases. *AJR Am J Roentgenol* 1994;162(4):919–22.
86. Kitagawa Y, Ito H, Amano Y, et al. MR imaging for preoperative diagnosis and assessment of local tumor extent on localized giant cell tumor of tendon sheath. *Skeletal Radiol* 2003;32(11):633–8.
87. Huang GS, Lee CH, Chan WP, et al. Localized nodular synovitis of the knee: MR imaging appearance and clinical correlates in 21 patients. *AJR Am J Roentgenol* 2003;181(2):539–43.
88. Fraire AE, Fechner RE. Intra-articular localized nodular synovitis of the knee. *Arch Pathol* 1972;93(5):473–6.
89. Rao AS, Vigorita VJ. Pigmented villonodular synovitis (giant-cell tumor of the tendon sheath and

- synovial membrane). A review of eighty-one cases. *J Bone Joint Surg Am* 1984;66(1):76–94.
90. Al-Nakshabandi NA, Ryan AG, Choudur H, et al. Pigmented villonodular synovitis. *Clin Radiol* 2004;59(5):414–20.
 91. Dorwart RH, Genant HK, Johnston WH, et al. Pigmented villonodular synovitis of the shoulder: radiologic-pathologic assessment. *AJR Am J Roentgenol* 1984;143(4):886–8.
 92. Dorwart RH, Genant HK, Johnston WH, et al. Pigmented villonodular synovitis of synovial joints: clinical, pathologic, and radiologic features. *AJR Am J Roentgenol* 1984;143(4):877–85.
 93. Pimpalnerkar A, Barton E, Sibly TF. Pigmented villonodular synovitis of the elbow. *J Shoulder Elbow Surg* 1998;7(1):71–5.
 94. Cotten A, Flipo RM, Chastanet P, et al. Pigmented villonodular synovitis of the hip: review of radiographic features in 58 patients. *Skeletal Radiol* 1995;24(1):1–6.
 95. Byers PD, Cotton RE, Deacon OW, et al. The diagnosis and treatment of pigmented villonodular synovitis. *J Bone Joint Surg Br* 1968;50(2):290–305.
 96. Hughes TH, Sartoris DJ, Schweitzer ME, et al. Pigmented villonodular synovitis: MRI characteristics. *Skeletal Radiol* 1995;24(1):7–12.
 97. Kottal RA, Vogler JB 3rd, Matamoros A, et al. Pigmented villonodular synovitis: a report of MR imaging in two cases. *Radiology* 1987;163(2):551–3.
 98. Spence LD, Adams J, Gibbons D, et al. Rice body formation in bicipito-radial bursitis: ultrasound, CT, and MRI findings. *Skeletal Radiol* 1998;27(1):30–2.
 99. Dale K, Smith HJ, Paus AC, et al. Dynamic MR-imaging in the diagnosis of pigmented villonodular synovitis of the knee. *Scand J Rheumatol* 2000;29(5):336–9.
 100. Jamieson TW, Curran JJ, Desmet AA, et al. Bilateral pigmented villonodular synovitis of the wrists. *Orthop Rev* 1990;19(5):432–6.
 101. Jelinek JS, Kransdorf MJ, Utz JA, et al. Imaging of pigmented villonodular synovitis with emphasis on MR imaging. *AJR Am J Roentgenol* 1989;152(2):337–42.
 102. Weiss SW, Goldblum JR, Enzinger FM. Benign tumors of peripheral nerves. In: Weiss SW, Goldblum JR, editors. *Enzinger and Weiss' soft tissue tumors*. 5th edition. Philadelphia: Mosby Elsevier; 2008. p. 825–901.
 103. Miettinen M. Diagnostic soft tissue pathology. New York: Churchill Livingstone; 2003.
 104. Sheikh S, Gomes M, Montgomery E. Multiple plexiform schwannomas in a patient with neurofibromatosis. *J Thorac Cardiovasc Surg* 1998;115(1):240–2.
 105. Ogoose A, Hotta T, Morita T, et al. Multiple schwannomas in the peripheral nerves. *J Bone Joint Surg Br* 1998;80(4):657–61.
 106. Kehoe NJ, Reid RP, Semple JC. Solitary benign peripheral-nerve tumours. Review of 32 years' experience. *J Bone Joint Surg Br* 1995;77(3):497–500.
 107. Brasfield RD, Das Gupta TK. Von Recklinghausen's disease: a clinicopathological study. *Ann Surg* 1972;175(1):86–104.
 108. Jett K, Friedman JM. Clinical and genetic aspects of neurofibromatosis 1. *Genet Med* 2010;12(1):1–11.
 109. Evans DG, Baser ME, McCaughran J, et al. Malignant peripheral nerve sheath tumours in neurofibromatosis 1. *J Med Genet* 2002;39(5):311–4.
 110. Li MH, Holtas S. MR imaging of spinal neurofibromatosis. *Acta Radiol* 1991;32(4):279–85.
 111. Banks KP. The target sign: extremity. *Radiology* 2005;234(3):899–900.
 112. Jee WH, Oh SN, McCauley T, et al. Extraaxial neurofibromas versus neurilemmomas: discrimination with MRI. *AJR Am J Roentgenol* 2004;183(3):629–33.
 113. Cohen LM, Schwartz AM, Rockoff SD. Benign schwannomas: pathologic basis for CT inhomogeneities. *AJR Am J Roentgenol* 1986;147(1):141–3.
 114. Peh WC, Shek TW, Yip DK. Magnetic resonance imaging of subcutaneous diffuse neurofibroma. *Br J Radiol* 1997;70(839):1180–3.
 115. Morton TS. VI. Metatarsalgia (Morton's painful affection of the foot), with an account of six cases cured by operation. *Ann Surg* 1893;17(6):680–99.
 116. Wu KK. Morton's interdigital neuroma: a clinical review of its etiology, treatment, and results. *J Foot Ankle Surg* 1996;35(2):112–9 [discussion: 187–8].
 117. Bencardino J, Rosenberg ZS, Beltran J, et al. Morton's neuroma: is it always symptomatic? *AJR Am J Roentgenol* 2000;175(3):649–53.
 118. Zanetti M, Strehle JK, Zollinger H, et al. Morton neuroma and fluid in the intermetatarsal bursae on MR images of 70 asymptomatic volunteers. *Radiology* 1997;203(2):516–20.
 119. Williams JW, Meaney J, Whitehouse GH, et al. MRI in the investigation of Morton's neuroma: which sequences? *Clin Radiol* 1997;52(1):46–9.
 120. Zanetti M, Weishaupt D. MR imaging of the forefoot: Morton neuroma and differential diagnoses. *Semin Musculoskelet Radiol* 2005;9(3):175–86.
 121. Erickson SJ, Canale PB, Carrera GF, et al. Interdigital (Morton) neuroma: high-resolution MR imaging with a solenoid coil. *Radiology* 1991;181(3):833–6.
 122. Weishaupt D, Treiber K, Kundert HP, et al. Morton neuroma: MR imaging in prone, supine, and upright weight-bearing body positions. *Radiology* 2003;226(3):849–56.
 123. Weiss SW, Goldblum JR, Enzinger FM. Benign tumors and tumor-like lesions of blood vessels. In: Weiss SW, Goldblum JR, editors. *Enzinger and Weiss' soft tissue tumors*. 5th edition. Philadelphia: Mosby Elsevier; 2008. p. 633–702.

124. Mulliken JB, Glowacki J. Hemangiomas and vascular malformations in infants and children: a classification based on endothelial characteristics. *Plast Reconstr Surg* 1982;69(3):412–22.
125. Mulliken JB, Young AE. *Vascular birthmarks: hemangiomas and malformations*. Philadelphia: Saunders; 1988.
126. Kransdorf MJ, Murphey MD, Fanburg-Smith JC. Classification of benign vascular lesions: history, current nomenclature, and suggestions for imagers. *Am J Roentgenol* 2011;197:1–4.
127. Ly JQ, Sanders TG, Mulloy JP, et al. Osseous change adjacent to soft-tissue hemangiomas of the extremities: correlation with lesion size and proximity to bone. *AJR Am J Roentgenol* 2003;180(6):1695–700.
128. Baek HJ, Lee SJ, Cho KH, et al. Subungual tumors: clinicopathologic correlation with US and MR imaging findings. *Radiographics* 2010;30(6):1621–36.
129. Shugart RR, Soule EH, Johnson EW Jr. Glomus tumor. *Surg Gynecol Obstet* 1963;117:334–40.
130. Sawada S, Honda M, Kamide R, et al. Three cases of subungual glomus tumors with von Recklinghausen neurofibromatosis. *J Am Acad Dermatol* 1995;32(2 Pt 1):277–8.
131. Okada O, Demitsu T, Manabe M, et al. A case of multiple subungual glomus tumors associated with neurofibromatosis type 1. *J Dermatol* 1999;26(8):535–7.
132. Park EA, Hong SH, Choi JY, et al. Glomangiomas: magnetic resonance imaging findings in three cases. *Skeletal Radiol* 2005;34(2):108–11.
133. Weiss SW, Goldblum JR, Enzinger FM. Perivascular tumors. In: Weiss SW, Goldblum JR, editors. *Enzinger and Weiss' soft tissue tumors*. 5th edition. Philadelphia: Mosby Elsevier; 2008. p. 751–67.
134. Drape JL, Idy-Peretti I, Goettmann S, et al. Subungual glomus tumors: evaluation with MR imaging. *Radiology* 1995;195(2):507–15.
135. Kneeland JB, Middleton WD, Matloub HS, et al. High resolution MR imaging of glomus tumor. *J Comput Assist Tomogr* 1987;11(2):351–2.
136. Matloub HS, Muoneke VN, Prevel CD, et al. Glomus tumor imaging: use of MRI for localization of occult lesions. *J Hand Surg Am* 1992;17(3):472–5.
137. Tachibana R, Hatori M, Hosaka M, et al. Glomus tumors with cystic changes around the ankle. *Arch Orthop Trauma Surg* 2001;121(9):540–3.
138. Stout AP. Myxoma, the tumor of primitive mesenchyme. *Ann Surg* 1948;127(4):706–19.
139. Weiss SW, Goldblum JR, Enzinger FM. *Enzinger and Weiss' soft tissue tumors*. 5th edition. Philadelphia: Mosby Elsevier; 2008.
140. Kransdorf MJ, Murphey MD. Diagnosis please. Case 12: Mazabraud syndrome. *Radiology* 1999;212(1):129–32.
141. Enzinger FM. Intramuscular myxoma; a review and follow-up study of 34 cases. *Am J Clin Pathol* 1965;43:104–13.
142. Murphey MD, McRae GA, Fanburg-Smith JC, et al. Imaging of soft-tissue myxoma with emphasis on CT and MR and comparison of radiologic and pathologic findings. *Radiology* 2002;225(1):215–24.
143. Bancroft LW, Kransdorf MJ, Menke DM, et al. Intramuscular myxoma: characteristic MR imaging features. *AJR Am J Roentgenol* 2002;178(5):1255–9.
144. Luna A, Martinez S, Bossen E. Magnetic resonance imaging of intramuscular myxoma with histological comparison and a review of the literature. *Skeletal Radiol* 2005;34(1):19–28.
145. Iwasko N, Steinbach LS, Disler D, et al. Imaging findings in Mazabraud's syndrome: seven new cases. *Skeletal Radiol* 2002;31(2):81–7.