Pre-procedural Imaging

15

Alexander Ghatan, Ian D. Dworkin, and George C. Chang Chien

Introduction

Regenerative medicine presents exciting new opportunities in the treatment of a variety of musculoskeletal (MSK) disorders; however, proper pre-procedural workup cannot be overlooked and must be completed prior to the initiation of such treatments. Pre-procedural imaging is crucial in both identifying the pathology that can be targeted by various regenerative techniques and ruling out pathology that will not benefit from treatment options. Additionally, pre-procedural imaging will help identify contraindications to regenerative treatments and evaluate for any "red flag" pathology. Conventional radiography has traditionally been helpful at identifying pathology; however, there are many MSK disorders that cannot be properly evaluated early enough with these modalities when regenerative therapies can provide the greatest benefit [1].

Patient Factors and Selection

Though there are no commonly accepted guidelines specific to regenerative medicine injections, there are such factors that are commonly evaluated before conducting conventional interventional procedures such as epidurals. For these spinal procedures, the ideal time to discontinue anticoagulation agents such as Coumadin, clopidogrel, and aspirin is unique to the pharmacokinetics of each individual medication; however, the North American Spine Society (NASS) recommends that an interval of approximately 1 week prior to surgery is prudent [2]. The risks involved in holding anticoagulation are also unique to each patient and must be weighed against the potential benefits of the treatment being provided. Contraindications for steroid injections have been well described in the literature, but there is little evidence for any particular contraindications for regenerative techniques. Table 15.1 lists several common contraindications and patient pre-procedure recommendations that many clinicians use to guide injection candidacy.

While regenerative medicine has an enormous capacity for healing various MSK disorders, it is important to recognize regenerative medicine's limitations and select patients and pathology that will best respond to these various techniques. Regenerative medicine is generally most effective for mild-to-moderate disease, including osteoarthritis Kellgren–Lawrence grade 1 or 2 or grade 1 or 2 ligamentous sprains (discussed below). Surgical management may be more appropriate for complete tears or end-stage, grade 4 osteoarthritis, and thus, pre-procedural imaging can assist in this patient selection. Additional patient characteristics that would impair the body's ability to heal or degrade its regenerative capacity include smoking cigarettes, uncontrolled blood glucose, immunosuppressed states, or active infections. Areas that lack adequate blood supply, such as eschars, or dysvascular or necrotic limbs, are also unlikely to respond to regenerative medicine techniques given their poor capacity to receive and utilize the necessary nutrients for repair.

Common Soft Tissue Injuries (Sprains/Strains)

Many common soft tissue injuries can be treated using regenerative techniques, and therefore, accurately localizing, identifying, and quantifying various injuries are valuable in any clinical setting. Imaging will help localize pathology, but a thorough history and physical examination are necessary in deciding what imaging to obtain. Every patient presents differently,

A. Ghatan (✉)
UCLA/VA of Greater Los Angeles, Department of Physical Medicine and Rehabilitation, Los Angeles, CA, USA

I. D. Dworkin
Board Certified in Physical Medicine and Rehabilitation and Pain Medicine, Department of Interventional Pain Physiatry, Newport Care Medical Group, Newport Beach, CA, USA

G. C. Chang Chien
GCC Institute, Department of Musculoskeletal Medicine and Medical Aesthetics, Newport Beach, CA, USA

C. W Hunter et al. (eds.), *Regenerative Medicine*, https://doi.org/10.1007/978-3-030-75517-1_15

Table 15.1 Pre-procedure patient preparation [2]

Strong Recommendations
Avoid NSAIDs and other anti-inflammatory agents for at least 7 days prior to Platelet Rich Plasma
No eating or drinking 6 hours before the procedure.
Patients are encouraged to hydrate well the day before the procedure.
Patients are encouraged to shower in the morning prior to their procedure.
Patients are advised to avoid using any products on their skin (lotion, makeup, sprays, anything topical to the area) the day of the procedure.
Relative Contraindications
Fever
Cancer
Rash over injection site
Elevated INR or actively taking anticoagulants
Poorly Controlled Type II Diabetes Mellitus or Elevated hemoglobin A1C

and thus, it is important for a clinician to be able to properly evaluate and describe various injuries in a standardized fashion. Below, we briefly discuss common terminology used in describing sprains, strains, and other common soft tissue injuries.

Tendinopathy

Tendinopathies are the various conditions associated with tendon pain caused by overuse. Tendinopathy is associated with histopathologic changes such as minimal inflammation, degeneration and disorganization of collagen fibers, and increased cellularity [3, 4]. Macroscopic changes include pain, tendon thickening, and the loss of mechanic [4]. Some suggest that tendon overuse leads to an imbalance between the protective/regenerative changes of the tissue, and pathologic responses from overuse, which results in pain, tearing, weakness, and degeneration [5].

Tendon and ligament abnormalities are widely assessed by MRI and ultrasound. The high levels of type I collagen in healthy tendons and ligaments, arranged in a cross-linked triple-helix structure, coupled with a structured orientation, provide their characteristic imaging appearances as well as cause particular imaging artifacts on various imaging modalities [6]. Tendons that pass through tight tunnels or around corners are typically covered in a tendon sheath, which is comprised of 2 layers of synovium. Otherwise, tendons are covered by a thin layer of loose fatty connective tissue called the paratenon [6]. The orientation of a tendon's fibers depends on the tension to which the tendon is subjected [7]. For tendons in which the force is directed along the tendon, the collagen is typically aligned along the tendon's long axis. Some tendons have a more complex structure with fibers running in discrete bundles. This is the case for tendons with origins from more than one muscle, such as the quadriceps tendon and the Achilles tendon (Fig. 15.1) [6].

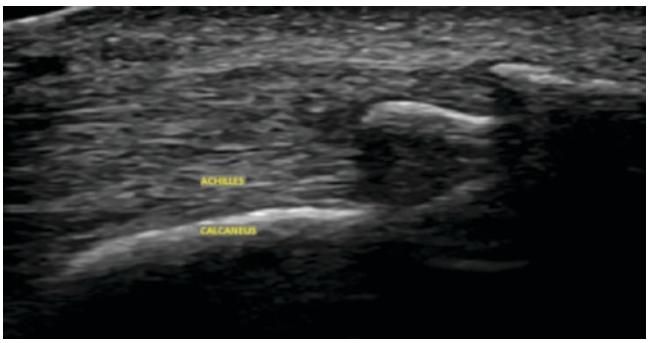

Fig. 15.1 Long axis ultrasound view of chronic Achilles tendonitis with enthesophyte irregularity and calcifications at the Achilles tendon insertion. (Reproduced from Benjamin et al. [61])

With age, changes in collagen structure such as a loss of water content predispose them to damage [8]. Vascularity also decreases with age, and tendon disease often occurs at these hypovascular areas. Instability or impingement leads to abnormal and excessive loading of the tendon which predisposes to injury [9, 10]. Collagen fibrils can rupture, and these regions may together form intrasubstance tears. These intrasubstance tears may extend to the surface, eventually progressing to full-thickness tears [9, 10]. Though ingrowth of vessels into the tendon is common, there is no evidence of inflammatory mediators [11–14]. Generally, degenerative changes occur before macroscopic tendon tears develop, and as such, it is unusual for a tear to occur in a nondegenerated tendon [6].

Ligament Sprains

Though ligaments are functionally different from tendons as they connect bone to bone, they are structurally similar [6]. The main differences are that ligaments have higher proteoglycan content, higher water content, lower in collagen content, and are less uniform [15]. An additional feature of ligamentous injuries is that because ligaments guide movement at joints, injury is typically associated with joint derangement.

Acute trauma typically causes ligament abnormalities and is often marked by fluid surrounding the ligament, although chronic repetitive microtrauma may be a factor as with tendon injuries [16, 17]. Potential damage includes interstitial tearing of collagen fibers and partial tears that extend to the surface and full-thickness ligament ruptures. Over time, the ligament can become elongated and lax. Other evidence of injuries includes bone contusions, fractures, or joint effusion. After healing, the ligament may appear thickened, weakened, and prone to further damage [6].

Table 15.2 describes the American Academy of Orthopedic Surgeons classification of ligamentous sprains [18]. Each

Table 15.2 AAOS classification of ligamentous sprains [18]

Grade	Description
Grade 1: Mild sprain	Typically described as stretching of the fibrils which may include microscopic damage and swelling, but the gross integrity of the ligament is usually not compromised.
Grade 2: Moderate sprain	Involves partial tearing of the ligament, which can result in laxity
Grade 3: Severe sprain	Complete tear of the ligament usually resulting in instability and interferes with joint function.

grade is based on the extent to which the ligament fibrils are interrupted and damaged. Of note, grade 3 injuries also include avulsion injuries, where a piece of the bone is pulled off along with the ligament.

Muscle Injuries

A strain is defined as an injury to the muscle and/or tendon, commonly at the musculotendinous junction [18]. Similar to sprains, strains are graded on a continuum. There can be a mild stretch injury with microscopic damage to the muscle fibers, or the injury can be more severe with partial or complete tear of the muscle–tendon complex. Chronic sprains and strains are common sources of pain. Patients may present with chronic pain, weakness, pain-limited range of motion (ROM), muscle spasms, muscle weakness, edema, or cramping. Repetitive strains and sprains can lead to further functional loss and can be a major pain generator that can be targeted with regenerative medicine.

When an indirect muscle injury occurs, there is a sudden onset muscle pain. It is usually localized to a single muscle and often occurs during an eccentric muscle contraction. The most commonly strained muscles in athletes are the biceps femoris, rectus femoris, and medial gastrocnemius [19]. Muscle strain grading systems can be based on function or imaging which will be discussed in later sections of this chapter and in the ultrasound chapter. Strains can be classified based on the amount functional loss from the patient's baseline (Table 15.3). Of note, grade 3 injuries are the rarest type of muscle injuries and often require surgical intervention. Avulsion injuries are occasionally described as Grade 3b muscle strain injuries [19].

Please see the chapters on MRI and ultrasound for additional information regarding muscle strain grading systems based on these modalities. MRI and ultrasound will also be further reviewed below. Unlike bone, muscles have a limited capacity for muscle regeneration and the majority of healing is by scar formation [19]. Thus, old or chronic muscle injuries may appear like an area of scar tissue within the normal-appearing muscle.

Table 15.3 Classification of muscle strains based on functional loss

Grade	Description
Grade 1: Mild	Stretch injury which results in less than 5% functional loss
Grade 2: Moderate	Partial muscle tear with 5–50% loss of function
Grade 3: Severe	Near-complete to complete rupture where there is greater than 50% loss of function. Typically seen at musculotendinous junction with a hematoma filling the space between the two ends.

Pre-Procedural Imaging and Common Imaging Findings

X-Ray and Computed Tomography (Ct)

For most musculoskeletal conditions, X-ray is often the first imaging used, but when it comes to regenerative treatments, the utility of X-ray is limited. Plain radiographs are useful at identifying gross deformity, fracture, dislocation, severe osteoarthritis, and ruling out osteoarthritis vs. adhesive capsulitis [20]. It is also useful in assessing joint space narrowing seen in osteoarthritis, and the severity of disease is commonly described using Kellgren–Lawrence classification.

The Kellgren–Lawrence classification of osteoarthritis, or KL grading, uses 4 grades of classification (Table 15.4) [21]. This classification system was originally described using AP views of knee radiographs but is commonly used to describe osteoarthritis in other joints as well (Fig. 15.2).

There are several limitations in using KL grading. One limitation is that the system assumes a linear progression of disease, which is often not the case. A second limitation is that there are times when patients may have osteophyte formation and/or sclerosis without joint space narrowing. Third, if a patient has joint space narrowing without any osteophytes, the KL grading system cannot be applied. X-ray fluoroscopy is also important in evaluating intervertebral disk integrity during diskography. Please see the following section for more information regarding diskography.

Computed tomography (CT) scans provide detailed visualization of bony structures and may assist in visualizing fractures not visible on X-ray [22]. They are furthermore readily available and quickly obtainable if the patient is unable to have an MRI; however, X-ray and CT are not typically used in imaging soft tissue injuries as they provide little insight into soft tissue pathology vital to pre-regenerative medicine procedures.

Magnetic Resonance Imaging (MRI)

When it comes to regenerative medicine, healing and repairing soft tissue are paramount, and therefore, the best imaging modality of soft tissues is with MRI. In this section, we will discuss the basics of how MRIs work, the different types of MRI, and some common pathological soft tissue findings that may be targeted with regenerative medicine.

Table 15.4 Kellgren–Lawrence classification of osteoarthritis [21]

Grade	Description
Grade 1	Doubtful narrowing of joint space and possible osteophyte formation.
Grade 2	Definite osteophytes and possible narrowing of joint space.
Grade 3	Moderate/multiple osteophyte formation, definite narrowing of joints space, some sclerosis, and possible deformity of bone contour.
Grade 4	Large osteophytes, severe narrowing of joint space, severe sclerosis, and definite deformity of bone contour are apparent.

The basis of MRI is in the magnetic resonance of hydrogen protons within the tissue being imaged [22]. Hydrogen protons, similar to tiny magnets with north and south poles, are susceptible to external magnetic fields. When hydrogen protons enter a strong external magnetic field, like an MRI scanner, most of the protons will align themselves in parallel to the strong field. An additional magnetic field, called a gradient, can be manually added to the MRI's native magnetic field, which creates an additional subdivision in the total magnetic field. The protons can then be triggered to flip or spin by radio-frequent pulses with a specific frequency. This causes the hydrogen protons to spin simultaneously, shifting/flipping back and forth in different axis, and is termed excitation and relaxation. Eventually, these induced magnetic fields/signal changes are registered by receiver coils and processed into the MRI image on a gray scale based on signal intensity. High signal intensity is seen as white, intermediate signal intensity appears gray, while low signal intensity appears dark gray or black.

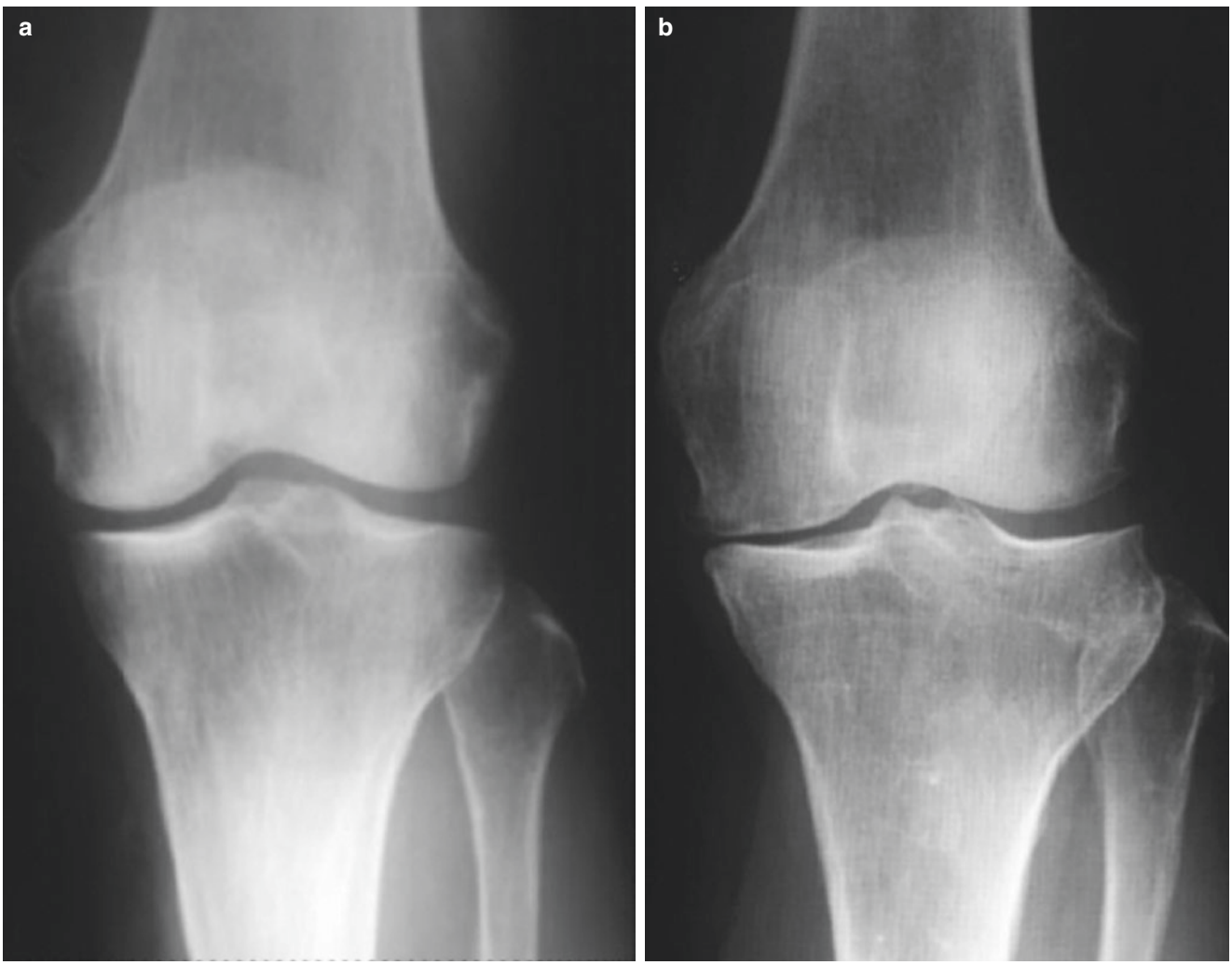

Fig. 15.2 AP plain X-rays of Kellgren–Lawrence grade 1 (**a**) and grade 2 (**b**). (Reproduced from Akira Horikawa et al. [62])

Table 15.5 T1-Weighted MRI sequences [22]

Very low signal intensity (black)	Low signal intensity (dark gray)	Intermediate signal intensity (light gray)	High signal intensity (white)
Calcium Dense Cortical Bone Intravascular/Flowing Blood Air	Water CSF Collagen Cartilage Tendons Ligaments Scars Bone Marrow Edema	Protein Dense Tissue Abscesses/Cysts Normal Synovial Fluid	Fat Normal Bone Marrow Blood (static) Contrast (Gadolinium)

Table 15.6 T2Weighted MRI sequences. [22]

Very low signal intensity (black)	Low signal intensity (dark gray)	Intermediate signal intensity (light gray)	High signal intensity (white)
Calcium Dense Cortical one Intravascular/ Flowing Blood Air	Cartilage Tendons Ligaments	Cartilage Fat Muscles	Fluid Edema CSF

MRI Sequences

Individual MRI sequences are based on the combinations of various radio-frequent pulses and gradients which allow visualization of varying pathology [22].

T1-Weighted Sequences The most common use of T1-weighted imaging is in the visualization of normal musculoskeletal anatomy [22]. In this sequence, the image is determined by the differences in relaxation times between water and fat. Fat has a high signal intensity (white), and water has a low signal intensity (black). This is because in a T1 series, fat has a shorter relaxation time than water. Table 15.5 describes the expected signals for various anatomical structures.

T2-Weighted Sequences On T2-weighted images, water has high signal intensity (white) which makes it useful to highlight the edema and inflammation associated with pathology (Table 15.6). In T2, similar to T1, air and calcifications have very low signal intensity (dark) [22]. Fig. 15.3 demonstrates the differences between T1 and T2 MRI sequences.

Proton Density (PD)-Weighted Imaging Proton density-weighted imaging is a visual representation of protons per volume within tissue [22]. Tissues with lower proton density will have a low signal intensity and will appear dark. Tissues with higher proton density will have a high signal and appear white. Fat, being a proton-dense tissue, has a relatively high signal intensity (light gray) but not as high as in a T1-weighted image (white). Fluid has intermediate signal intensity rather than the high signal intensity seen on T2-weighted images.

A common use for PD-weighted imaging is in the evaluation of meniscal tears of the knee. PD-weighted imaging is also useful in distinguishing between CSF and pathology [22]. On T2-weighted imaging, CSF and many pathologies have a high signal but on PD-weighted imaging, the contrast between CSF (intermediate signal intensity) and most pathologies (high signal intensity) will be better visualized.

Fat Suppression imaging (STIR and SPIR) The suppression of adipose tissues is an option that can be used in various MRI sequences. Fat suppression images are commonly referred to as fat saturation images or "FatSat." This creates a low-signal intensity of fat which helps in contrasting it from vessels and various pathologies [22]. In musculoskeletal imaging, fat suppression can be useful. For example, bone marrow is high in fat which may mask bone barrow edema on a T2-weighted image. Thus, in suppressing the fat, edema from a fracture, tumor, or other pathology will be more easily visualized.

Short-tau inversion recovery (STIR) and spectral presaturation inversion recovery (SPIR) sequences are the most commonly used fat suppression sequences and are both T2-weighted images [22]. STIR sequences are very useful in detecting bone marrow edema.

Diffusion-Weighted Imaging (DWI) Diffusion refers to the random movement of molecules within a substance. The diffusion behavior of hydrogen molecules is determined by different field strengths [22]. DWI is T2-weighted images. This type of MRI is commonly employed in the diagnosis of acute strokes but is not often employed in the evaluation of MSK disorders.

MRI Contrast

When an MRI is performed with contrast, it will typically rely on a T1-weighted image since use with T2-weighted imagines have little value due to the fact that both fluid/edema and contrast will have a high signal intensity and be generally indistinguishable [22]. The most commonly used contrast type for MRI is gadolinium. It reduces the T1 relaxation time of the protons that absorb the contrast, and thus, these protons will have higher (white) signal intensity.

Fig. 15.3 T1 and T2 MRI sequences demonstrating decreased disk signal at L4/L5. (Reproduced from Michael [63])

Common indications for MRI contrast include detecting various lesions (tumor, metastases, infection, abscess), characterization of lesions, especially in the viscera, imaging of vessels/vascular pathology, and imagining of intraarticular structures (MR arthrogram) [22].

Tendon and Ligaments on MRI

The structure of tendons determines their appearance on MRI. Due to the abundance and orientation of collagen and water molecules, normal tendons appear as dark (low signal intensity) on most MRI sequences, including T1- and T2-weighted sequences [22]. With injury, the fluid signal within a tendon or ligament tears can be identified with *T*2-weighted images [10]. MRI provides high spatial resolution of tendons and ligaments. There is a direct correlation between image resolution and the strength of the MRI's magnetic field - as the strength of the field increases, so does the resolution of the image. Therefore, an MRI with a stronger the magnetic field is much more likely to detect a partial-thickness tear [6, 23].

Tendinopathies and Ligamentous Sprains on MRI

One of the first signs of a tendon injury on MRI is an increase in signal intensity, which can be seen on T1-weighted images [6]. Additionally, the tendon may appear thickened. The appearance of a tendon tear varies with chronicity. In the more acute setting, T2-weighted or STIR images may show increased fluid signal within tendon tears [24, 25]. In an older tear, scarring within the defect can produce an intermediate signal. Increased signal on T2-weighted images with fat suppression is the best way to diagnose tears on MRI with the best specificity (Fig. 15.4).

Partial-thickness tears often heal with the defects being filled with fluid or granulation tissue [6]. The resulting tissue is weaker than the native tendon and can propagate into full-thickness tears. When the entire tendon is disrupted, the torn ends can retract, altering the normal/expected anatomy, making visualization difficult. When this occurs, the secondary signs of full-thickness tears such as muscle edema, atrophy, tendon contour irregularity, and/or retraction of the musculotendinous junction assist in making the diagnosis.

Ligamentous sprains appear similarly on MRI. In the acute setting, T2-weighted or STIR images may show increased fluid signal around the ligament and may appear thickened with increased signal within the ligament [6]. In an older tear, the ligament may appear irregular, thickened, or possibly thinned.

Muscle Contusions on MRI

The role of imaging in acute muscle injury has changed from merely confirming a clinical diagnosis to defining the precise location and extent of the injury. Being able to measure the

size and extent of soft tissue disruption assists in predicting outcome and determining treatment. When assessing muscle injury by MRI, either a STIR, fat sat PD-weighted, or fat sat T2-weighted sequence should be utilized [19]. T1 should be included when assessing for blood products or atrophy. It is always important to compare the T1 and STIR series in suspected areas of muscle injury as a focal area of fatty infiltration (which may be due to atrophy) may be misinterpreted as an intramuscular scar.

Contusions typically occur when there is a blunt force trauma to a muscle without disruption to the skin. On MRI imaging, the appearance of contusions depends upon the blood products and fluid characteristics within the lesion, which changes with time (Table 15.7) [19].

In the hyperacute stage (<24 hours) of the injury, the contusion causes edema and interstitial hemorrhage, which leads to the characteristic feather-like high signal within the muscle on fat-suppressed fluid sensitive sequences (i.e., STIR, fat sat PD-weighted, or fat sat T2-weighted) [19]. The feather-like appearance occurs due to the high signal of blood and edema spreading between the individual muscle fibers.

Fig. 15.4 Grade 1 tendinosis on T2-weighted fat-suppressed MRI. (Reproduced from Andrea et al. [64])

In the acute stage (24–48 hours) of the injury, the contusion appears as an irregular muscle laceration [19, 22]. Blood products may result in areas of faint high signal on T1-weighted images; however, the same imaging findings could be seen in a low-grade muscle strain.

In the subacute stage (48–72 hours) of the injury, the contusion becomes a more clearly defined fluid collection within the muscle [19, 22]. The muscle surrounding the site of injury remains diffusely high signal on fluid-sensitive sequences. Characteristics of a hematoma will change with time depending on the nature of the blood product within it based on metabolic breakdown.

As time passes, a hematoma will undergo fibrosis and calcification [19, 22]. Fibrosis of the hematoma margins will contract the lesion over time. Calcification can lead to weakening, making the muscle susceptible to repeat injury.

Muscle Strains on MRI

As previously discussed, a muscle strain is an indirect muscle injury, which often occurs during an eccentric muscle contraction. Muscle strains can be graded via MRI based on the extent of cross-sectional area of disruption of the muscle fascicles as compared to clinical grading which was discussed above based on functional impairment [19]. MRI assists in determining the extent of cross-sectional fiber disruption, which most commonly occurs at the musculotendinous junction.

- *Grade 1 Strain*: There is less than 5% disruption in the cross-sectional area of the muscle. On fluid-sensitive fat-suppressed sequences (i.e., T2-weighted fat sat), there is an increased signal at the site of injury due to the edema and blood products radiating from the injury site which produces the classic feather-type appearance within the muscle on MRI. Perifascial fluid may also be seen.
- *Grade 2 Strain*: There is at least 5% but less than 100% disruption in the cross-sectional area of the muscle causing distortion of the normal muscle architecture. This typically results in hematoma formation at the musculotendinous junction. The feathery-type muscle edema pattern as described in grade 1 injury may also be present. There may also be some laxity of the central tendon within the muscle.
- *Grade 3 Strain*: There is complete disruption of the muscle, typically at the musculotendinous junction with a

Table 15.7 Appearance of muscle contusions on MRI [19]

	Hyperacute (<4 hour)	Acute (4–6 hour)	Early subacute (6–72 hour)	Late subacute (72 hour to 4 weeks)	Chronic (>4 weeks)
T1 Signal Intensity	Intermediate	Intermediate	High	High	Low
T2 Signal Intensity	High	Low	Low	High	Low

hematoma filling the space between the two ends. Grade 3 injuries are the rarest type of muscle injuries and often require surgical intervention. Avulsion injuries are occasionally described as grade 3b muscle strain injuries.

Unlike bone, muscle has a limited capacity for regeneration following injury [19]. The majority of healing is by scar formation. Thus, old or chronic muscle injuries may appear like an area of scar tissue within the normal-appearing muscle. Figure 15.5 demonstrates a T2-weighted MRI of complete rupture of left distal biceps femoris tendon at the musculotendinous junction.

MRI of the Spine

Obtaining MRI images of the spine is crucial for detecting various pathologies as it gives detailed visualization of the soft tissue, and the various aforementioned sequences can help differentiate between different injuries and lesions. Spine degeneration, such as spondyloarthropathies and disk degeneration, can be best visualized using MRI which is why it is the preferred imaging modality in back pain; however, while MRI provides a good visual representation of the spine, it cannot definitively localize patient's pain. Thorough history, clinical exam, and the possible addition of electrodiagnostics in conjunction with the imaging are necessary. There are other provocative exams and invasive tests that can be used to help identify the patient's pain, some of which will be discussed further in this chapter.

Disk degeneration and diskogenic back pain are prime targets for treatment with regenerative techniques. Signal changes of the disk, vertebral endplates, and subchondral bone are seen on MRIs of degenerative spines and are strongly associated with low back pain [26]. These bone marrow and vertebral end place lesions were originally classified in 1988 by Modic et al. and are referred to as "Modic changes." [27, 28] In 1990, Miller further classified these imaging findings into what is now known as "modified Modic changes," and in 2001, Weishupt et al. further classified Modic changes into four degrees based on the percentage of vertebral height involvement in a mid-sagittal image of the spine (Table 15.8) [29, 30].

Relationship between Modic Changes and Lower Back Pain Despite this characterization of spinal changes, only a small proportion of pathology can be diagnosed with certainty based on a pathoanatomical entity alone [31]. There is increasing evidence though that demonstrates the prevalence of Modic changes, especially type 1, increases in people with nonspecific low back pain compared to people without low back pain [32–34]. Modic changes at L5/S1 and, especially

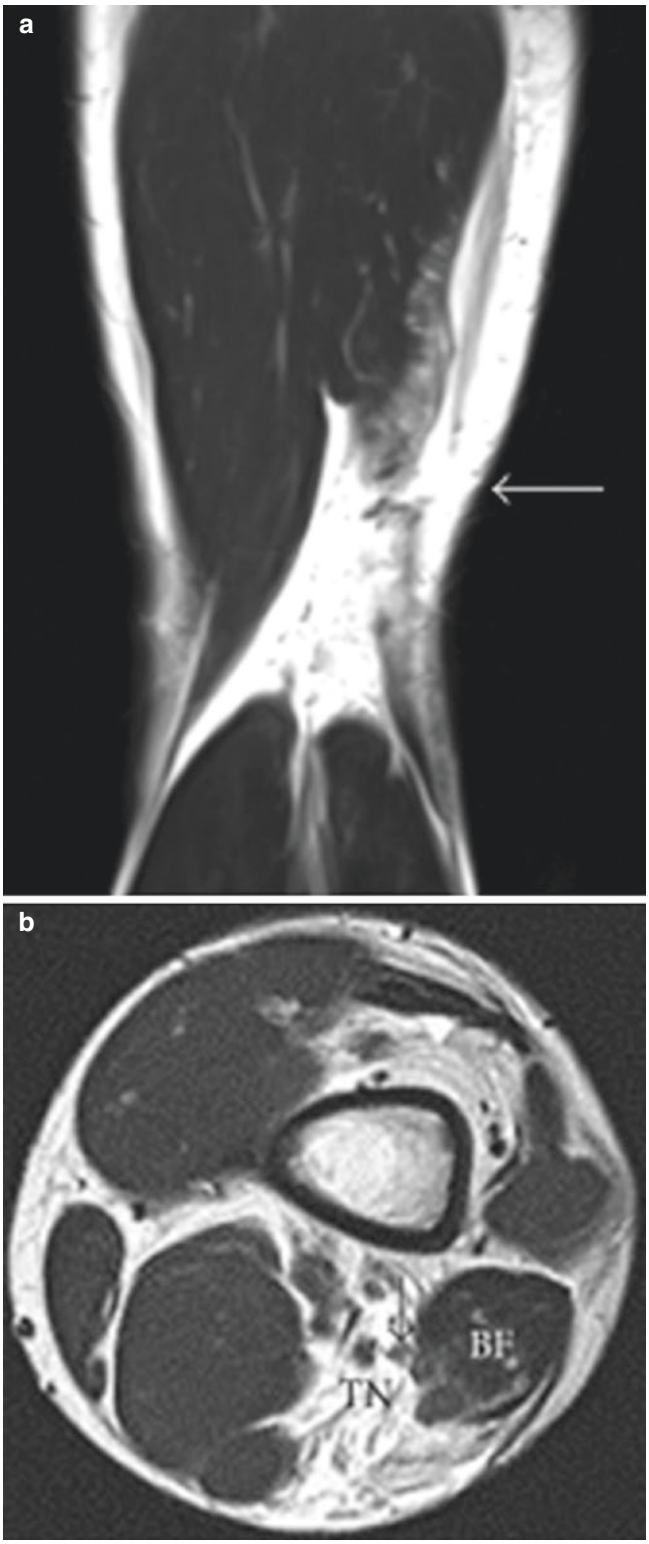

Fig. 15.5 T2-weighted MRI of a complete rupture of the distal biceps femoris tendon at the musculotendinous junction. (Reproduced from Aki Fukuda et al. [65])

Modic Type 1, are more likely related to low back pain than other levels and types of Modic changes (Fig. 15.6) [35]. Additionally, Modic changes are often associated with Schmorl's nodes, which occur when the nucleus pulposus herniates through the vertebral endplate and into the adjacent vertebral body (Fig. 15.7). On MRI, they appear as focal endplate defects (low signal on T1 and high signal on T2). They also have a well-defined herniation pit and a surrounding wall of high signal on T1 and T2 within the vertebral body [26, 36]. Though there is a lack of consensus regarding Schmorl's nodes clinical significance, Hamanishi et al. studied 400 patients with lower back pain and found that 19% of patients with back pain had Schmorl's nodes compared to only 9% of control patients [37].

Table 15.8 Modified modic changes combining Miller et al. and Weishupt et al. criteria [29, 30]

Modic Type	Description
Type 0 or first-degree changes	Normal; no degeneration. No MRI evidence of bone marrow or vertebral end plate lesions. No T1 or T2 changes
Type 1 or second-degree changes	Vertebral body and bone marrow edema/ inflammation and hypervascularity T1: low signal T2: high signal Mild signal intensity changes of less than or equal to 25% of the vertebral height
Type 2 or third-degree changes	Normal haemopoietic bone marrow is replaced by fat infiltration secondary to ischemia. T1: high signal T2: normal-appearing to high signal Moderate changes at 25–50% of the vertebral height
Type 3 or fourth-degree changes	Subchondral bony sclerosis seen T1: low signalT2: low signal Severe changes greater than 50% of the vertebral height

Differentiating Modic Changes from Spinal Infections and Tumors Spinal infections and tumors may appear similarly to Modic changes on MRI, but there are some important distinguishing characteristics [38]. Spondylodiskitis, an infection of the disk and vertebral body, presents as lesions with high signal on T2 compared to normal or low signal on T2 in disk degeneration. Spondylodiskitis can cause significant paravertebral soft tissue edema and can even lead to epidural mass effect [38, 39]. Erosion of vertebral body and end plates are always seen in intervertebral disk infections, whereas Modic changes may be focal or diffuse along the endplates, but tend to be linear and always parallel to the endplates [26, 40].

The most common type of neoplastic lesion found in the spinal column is secondary to metastasis [26]. Metastatic disk involvement is rare and is therefore easily distinguishable from Modic changes by the absence of disk space involvement.

Relationship between Modic Changes and Diskography Some authors report that when the signal

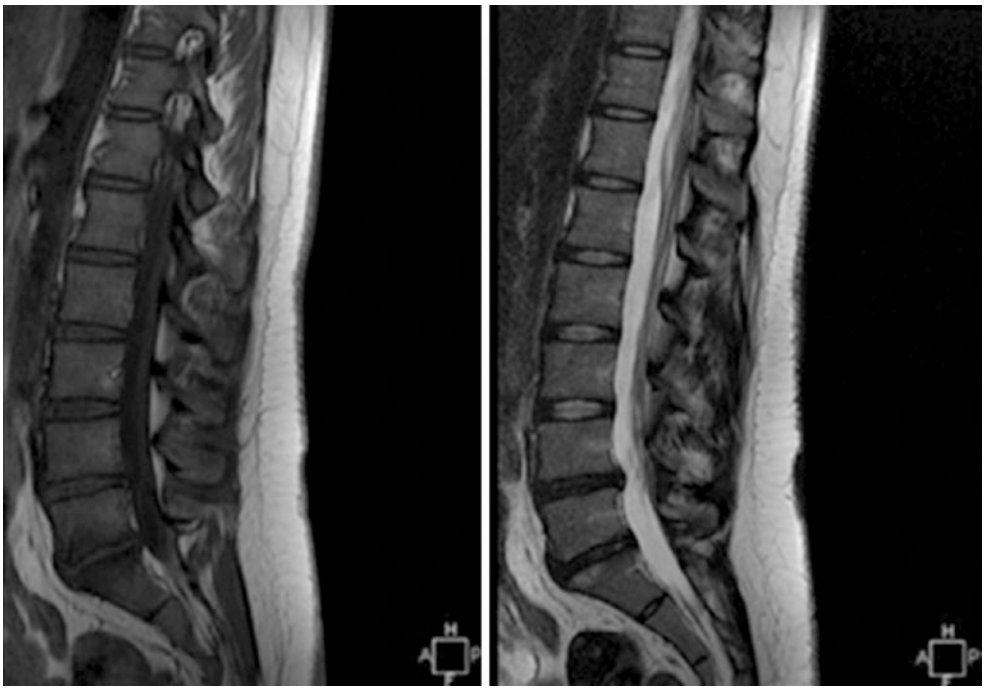

Fig. 15.6 Early reactive endplate changes at L5/S1 (Modic type 1). (Reproduced from Michael [63])

Fig. 15.7 Sagittal T2WI of a 17-year-old male with Scheuermann's disease with multilevel involvement of Schmorl's nodes and endplate irregularities. (Reproduced from Aikaterini et al. [66])

intensity changes in the endplates and decreased signal intensity in degenerative lumbar disks were combined, the specificity of using MRI to diagnose disk pain disease increases significantly [26, 41]. The signal intensity changes in endplates indicate a high degree of specificity, but lack sensitivity in diskogenic low back pain. Therefore, Modic changes are of important value in the diagnosis of diskogenic low back pain, but MRI does not completely replace the diskography due to the lack of the sensitivity. Diskography will be discussed in detail later in this chapter.

Relationship Between High-Intensity Zone on MRI and Diskography in Patients with Low Back Pain The presence of a high-intensity zone on MRI is another imaging finding that may indicate a patient's pain generator. The high-intensity zone (HIZ) was first described in lumbar spine MRI studies [42] and is defined as a focal area of high signal on T2-weighted sequences in the posterior annulus fibrosus. It has a considerably brighter signal intensity than nucleus pulposus from which it is distinctly disassociated [43–45].

The correlation between HIZ on MRI and diskography in patients with low back pain has been examined with varying results (Fig. 15.8).

Some data suggest that the presence of HIZ could be used as an indicator of annular tears and diskogenic low back pain [42, 43, 45–49]. Additionally, some authors posit that the HIZ is caused by the inflammation of annulus fibrosus and that there is a correlation between the presence of HIZ within the posterior annulus of a lumbar disk on MRI and the pain response following diskography in patients with low back pain. There is also evidence that HIZ is indicative of a Grade 3 to 4 annular tear and that the signal change is due to the accumulation of mucoid fluid within the fissure of the annulus. Others counter this, speculating that the value of HIZ is limited to the diagnosis of diskogenic low back pain [50–53]. Regardless, the finding of a HIZ should be investigated by diskography and potentially treated as a patient's pain generator.

MRI Limitations

The MRI is useful for lesion detection and localization; however, it is expensive, time-consuming, and can be uncomfortable, particularly for patients with claustrophobia [19]. It also only acquires static images. Additionally, MRI is contraindicated in patients with certain pacemakers and surgical brain clips.

Ultrasound

Ultrasound imaging has several advantages over MRI including superior spatial resolution, lower cost, patient and practitioner convenience, portability, and is essentially the only imaging modality that can provide dynamic imaging of musculoskeletal injuries and is a crucial tool in needle guidance of various joint injections (Fig. 15.9) [19]. One significant limitation of ultrasounds is operator dependency and the need for an acoustic window which can be difficult to obtain. Images can vary depending on the skill of technique, knowledge of anatomy, and experience. Ultrasound also has limited fields of vision and cannot penetrate bone. Additionally, injuries under ultrasound are less prominent/obvious than in MRI, which can also image both ligamentous injuries and associated intraarticular damage. Ultrasound basics will be reviewed here, but please refer to this text's chapter on ultrasound for additional, more comprehensive information.

Ultrasound Basics

Echogenicity is the ability of a tissue to reflect or transmit ultrasound waves in the context of surrounding tissue [54]. Hyperechoic tissue appears white, hypoechoic tissue appears gray, and anechoic tissue appears black. The following are the appearances of commonly evaluated structures under ultrasound:

- Bone appears anechoic (black) with a hyperechoic rim (bright) because the beam cannot penetrate bone; thus, it casts in acoustic shadow behind it.
- Cartilage is hypoechoic (gray) and is more penetrable than bone.
- Blood vessels appear anechoic (black) and can differentiate between veins and arteries as veins are easily collaps-

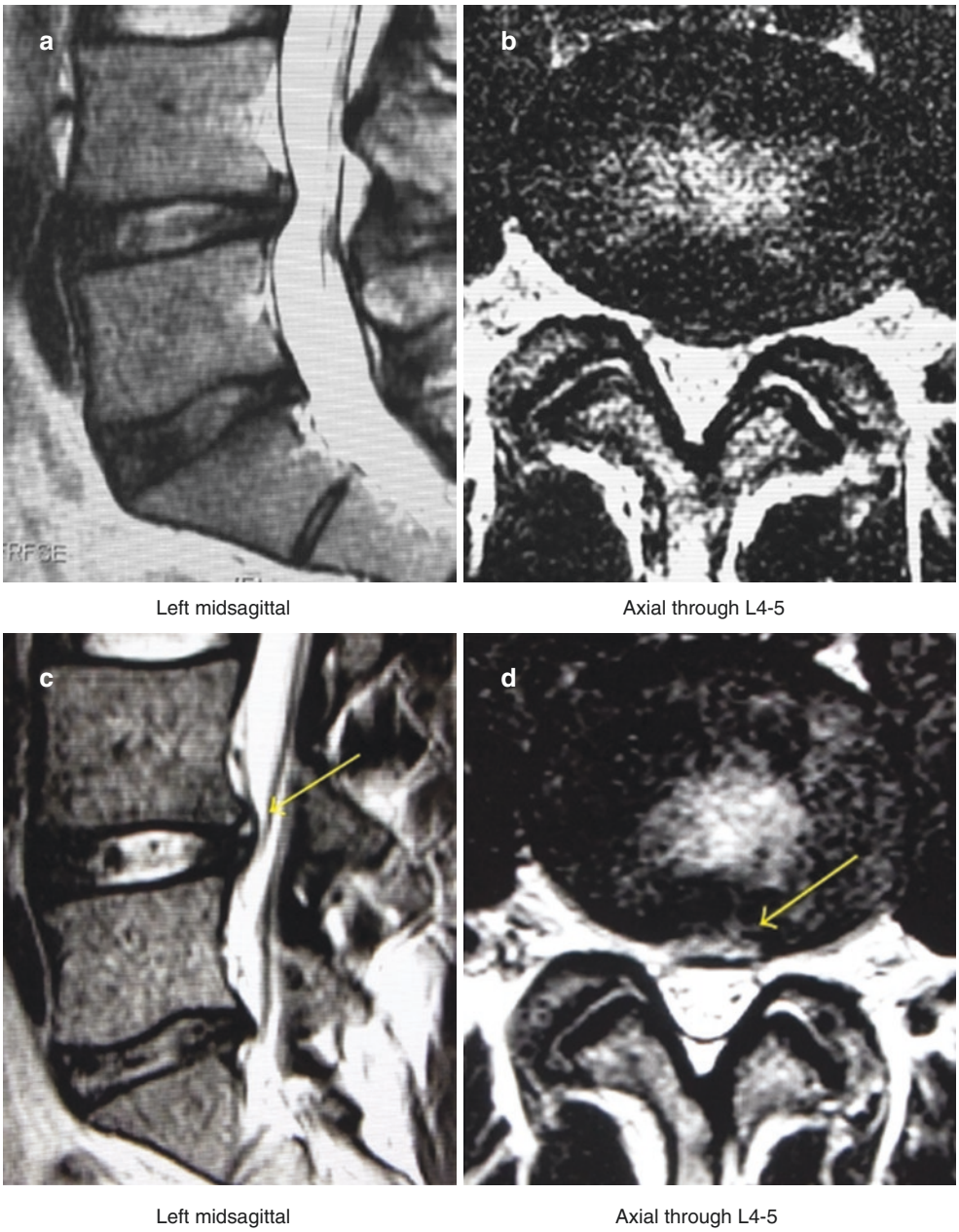

Fig. 15.8 Serial T2-weighted MRI findings of a degenerated disk with a slight protrusion is visible; however, originally, no high signal intensity zone (HIZ) is obvious. Subsequent imaging reveals obvious HIZ. (Reproduced from Kosuke et al. [67])

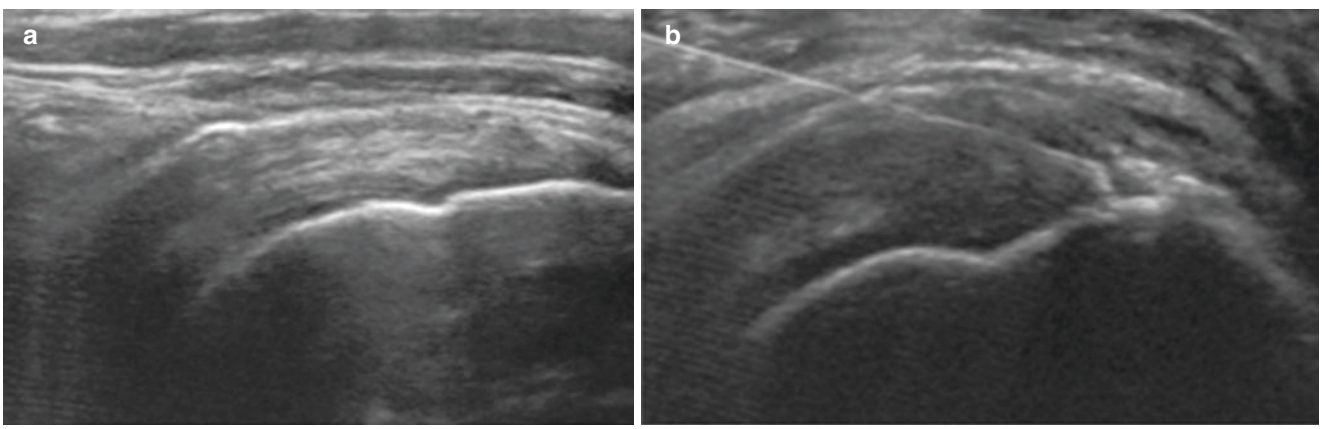

Fig. 15.9 US-guided Injection into the subacromial bursa (**a**) and supraspinatus tendon tear (**b**)

ible when pressure is applied by the transducer, while arteries are pulsatile and are not easily collapsible.
- Muscles are hypoechoic (gray) with striate structure.
- Fat is almost anechoic (black).
- Fascia/connective tissue strands/fascicles appear as hyperechoic (white) lines and bands.

Contusions on Ultrasound

Contusions on ultrasound are Ill-defined areas of hyperechogenicity within a muscle that crosses fascial boundaries [19]. They can be hyperacute, acute, or subacute.

If the contusion is *Hyperacute (<24 hours)*, the injured muscle appears swollen and may be isoechoic with adjacent normal-appearing muscle [19]. If the injury was from a forceful trauma, there may be significant rupture of muscle fibers and bleeding into the potential space resulting in a hematoma.

If the contusion is *acute (24–48 hours),* hematoma will appear as an irregularly outlined muscle laceration with hypoechoic fluid inside [19]. During this period, the hematoma may solidify and become hyperechoic compared to the surrounding muscle.

Finally, if the contusion is *subacute (48–72 hours)*, it becomes a clearly defined hypoechoic fluid collection with an echogenic margin [19]. Over time, this echogenic margin gradually enlarges and fills in the hematoma in a centripetal fashion.

If the hematoma is causing significant pain, exerting mass effect on neurovascular structures, or is placing the tissue at risk for compartment syndrome, clot evacuation may be considered via ultrasound guidance at 10–14 days after the initial injury [19].

Muscle Strain on Ultrasound

Muscle strains on ultrasound are rated on a three-point grading system as shown in Table 15.9 [55].

Tendons and Ligaments on Ultrasound

The tendon's fascicular structure is seen on ultrasound as closely spaced echogenic lines on longitudinal scanning. In the transverse plane, echogenic dots or lines are seen. While ligaments also appear as echogenic fibrillar structures [56], they are less echogenic than tendons [57] due to their less regular structure. The reflective fascicles within the tendons and ligaments can be seen best when the ultrasound beam is perpendicular to the fascicles' orientation and a different group of fibers can be seen by changing the probe orientation along the axis. Both tendons and ligaments exhibit anisotropy [6]. There is no echogenic appearance if the beam is not perpendicular which may simulate disease. This must be considered when examining tendons where the fibers change direction or are not parallel to the skin.

Table 15.9 Ultrasound grading of muscle strains

Grade	Characteristics
Grade 1	May appear normal or show areas of increased echogenicity at the injury site taking up less than 5% of the muscle substance in cross section. Long cavities within the muscle measuring 10 mm or less are also considered Grade 1.
Grade 2	>5% but <100% disruption of the cross-sectional area of the muscle typically visualized at the musculotendinous or myofascial junction.
Grade 3	Ultrasound shows complete disruption of the muscle at the musculotendinous junction. Surrounding muscle is hyperechoic, and intermuscular perifacial and subcutaneous fluid collections are commonly visualized.

Tendinopathy and Ligamentous Sprains on Ultrasound

Under ultrasound, tendinopathy appears as areas of less organized fibrillar structure with increased spacing between the hyperechoic fibrillar lines and overall reduced echogenicity, which are associated with tendon thickening [6]. The appearance of tendon tears depends on the chronicity of the injury. In the acute phase, there may be anechoic fluid within the tear, but with time the echogenicity can increase and the tendon may appear normal. Dynamic visualization can particularly aide in identifying tendon and ligamentous pathology that may otherwise be missed. Also, Doppler imaging is useful in helping distinguish between small intrasubstance tears and vessels that have developed within a tendinopathic tendon.

Under ultrasound, acute ligamentous sprains may appear as thickened areas of the ligament with diffuse hypoechogenicity and surrounding edema [58]. Ligamentous tears may appear as areas with reduced echogenicity that interrupt the ligament fibers [59]. An interruption that extends across the entire thickness of the tendon is considered a complete or full-thickness tear [6]. As healing progresses, the fluid surrounding the injury site dissipates but the thickening and the laxity on dynamic imaging may remain.

On ultrasound, tendinosis appears as heterogeneous areas with reduced echogenicity [60]. In more chronic tendinosis, there may be calcifications within the tendon with varying appearances.

Conclusion

Pre-procedural imaging is vital in the evaluation and diagnosing of various MSK diseases, as well as imperative to rule out other pathology that cannot be treated with regenerative techniques (cancer, abscesses, etc.). By understanding the different uses of X-ray, CT, MRI, and ultrasound, clinicians can choose the most appropriate imaging modality leading to more effective care. X-rays are often the first images obtained but have limited use outside of evaluating fractures and osteoarthritis. CT can provide more detailed visualization of bony structures, fluid collections, and can be used if MRI is

contraindicated but is also not typically employed to evaluate soft tissue injuries. MRI is the most important modality in pre-procedural imaging, but proper sequence selection and knowledge of their differences are crucial to their interpretation of underlying pathology. Diskography is an important modality to use for diskogenic pain if intradiskal stem cells are being considered because it is the gold standard in correlating imaging deficiencies with the patient's symptoms. Finally, ultrasound has quickly become a lynchpin of regenerative medicine, providing dynamic visualization of pathology and direct needle visualization to ensure the regenerative techniques reach their desired location. Most importantly though in pre-procedural preparation is the continued use of a thorough and well-documented history and physical examination which no imaging modality can supplant.

References

1. Tan AL, Wakefield RJ, Conaghan PG, Emery P, McGonagle D. Imaging of the musculoskeletal system: magnetic resonance imaging, ultrasonography and computed tomography. Best Pract Res Clin Rheumatol. 2003;17(3):513–28. https://doi.org/10.1016/S1521-6942(03)00021-4.
2. North American Spine Society. Evidence-based clinical guidelines for multidisciplinary spine care antithrombotic therapies in spine surgery. 2009. [Internet] 2018 Sep 1. Available from: https://www.spine.org/Documents/ResearchClinicalCare/Guidelines/AntithromboticTherapies.pdf.
3. Khan KM, Cook JL, Bonar F, Harcourt P, Astrom M. Histopathology of common tendinopathies. Update and implications for clinical management. Sports Med. 1999;27:393–408.
4. Soslowsky LJ, Thomopoulos S, Tun S, Flanagan CL, Keefer CC, Mastaw J, Carpenter JE. Neer award 1999. Overuse activity injures the supraspinatus tendon in an animal model: a histologic and biomechanical study. J Shoulder Elb Surg. 2000;9:79–84.
5. Andres BM, Murrell GAC. Treatment of tendinopathy: what works, what does not, and what is on the horizon. Clin Orthop Relat Res. 2008;466(7):1539–54. https://doi.org/10.1007/s11999-008-0260-1.
6. Hodgson RJ, O'Connor PJ, Grainger AJ. Tendon and ligament imaging. Br J Radiol. 2012;85(1016):1157–72. https://doi.org/10.1259/bjr/34786470.
7. O'Brien M. Anatomy of tendons. In: Maffulli N, Renstrom P, Leadbetter WB, editors. Tendon injuries. London: Springer-Verlag; 2005.
8. Tuite DJ, Renstrom PA, O'Brien M. The aging tendon. Scand J Med Sci Sports. 1997;7:72–7.
9. Pierre-Jerome C, Moncayo V, Terk MR. MRI of the Achilles tendon: a comprehensive review of the anatomy, biomechanics, and imaging of overuse tendinopathies. Acta Radiol. 2010;51:438–54.
10. Calleja M, Connell DA. The Achilles tendon. Semin Musculoskelet Radiol. 2010;14:307–22.
11. Kannus P, Jozsa L. Histopathological changes preceding spontaneous rupture of a tendon. A controlled study of 891 patients. J Bone Joint Surg Am. 1991;73:1507–25.
12. Aström M, Rausing A. Chronic Achilles tendinopathy. A survey of surgical and histopathologic findings. Clin Orthop Relat Res. 1995;316:151–64. PMID: 7634699.
13. Alfredson H, Thorsen K, Lorentzon R. In situ microdialysis in tendon tissue: high levels of glutamate, but not prostaglandin E2 in chronic Achilles tendon pain. Knee Surg Sports Traumatol Arthrosc. 1999;7:378–81.
14. Regan W, Wold LE, Coonrad R, Morrey BF. Microscopic histopathology of chronic refractory lateral epicondylitis. Am J Sports Med. 1992;20:746–9.
15. Simon SR. Orthopaedic basic science. Iowa: Am Acad Orthopaed Surg; 2000.
16. Bredella MA, Tirman PF, Fritz RC, Feller JF, Wischer TK, Genant HK. MR imaging findings of lateral ulnar collateral ligament abnormalities in patients with lateral epicondylitis. AJR Am J Roentgenol. 1999;173:1379–82.
17. Finlay K, Ferri M, Friedman L. Ultrasound of the elbow. Skelet Radiol. 2004;33:63–79.
18. American Academy of Orthopedic Surgeons. Sprains, strains and other soft tissue injuries [Internet] 2018 Sep 1. Available from: https://orthoinfo.aaos.org/en/diseases—conditions/sprains-strains-and-other-soft-tissue-injuries/.
19. Lee JC, Mitchell AWM, Healy JC. Imaging of muscle injury in the elite athlete. Br J Radiol. 2012;85(1016):1173–85. *PMC*. Web. 22 Sept. 2018.
20. O'Kane JW, Toresdahl BG. The evidenced-based shoulder evaluation. Curr Sports Med Rep. 2014;13:307–13. https://doi.org/10.1249/JSR.0000000000000090.
21. Kohn MD, Sassoon AA. Classifications in brief kellgren-lawrence classification of osteoarthritis. Clin Orthop Relat Res. 2016;74(8):1886–93.
22. Westbrook C, Roth C, Talbot J. MRI in practice. Chichester: Wiley-Blackwell; 2011.
23. Magee T, Williams D. 3.0-T MRI of the supraspinatus tendon. AJR Am J Roentgenol. 2006;187:881–6.
24. Stoller DW, Wolf EM, Li AE, Nottage WM, Tirman PFJ. The shoulder. In: Stoller DW, editor. Magnetic resonance imaging in orthopaedics and sports medicine. Baltimore: Lippincott Williams & Wilkins; 2007. p. 1131–462.
25. Reinus WR, Shady KL, Mirowitz SA, Totty WG. MR diagnosis of rotator cuff tears of the shoulder: value of using T_2-weighted fat-saturated images. AJR Am J Roentgenol. 1995;164:1451–5.
26. Zhang Y-H, et al. Modic Changes: A Systematic Review of the Literature. Eur Spine J. 2008;17(10):1289–99. *PMC*. Web. 22 Sept. 2018.
27. Modic MT, Masaryk TJ, Ross JS, Carter JR. Imaging of degenerative disk disease. Radiology. 1988;168:177–86.
28. Modic MT, Steinberg PM, Ross JS, Masaryk TJ, Carter JR. Degenerative disk disease: assessment of changes in vertebral body marrow with MR imaging. Radiology. 1988;166:193–9.
29. Miller G. The spine. In: Berquist T, editor. MRI of the musculoskeletal system. 2nd ed. New York: Raven; 1990.
30. Weishaupt D, Zanetti M, Hodler J, Min K, Fuchs B, Pfirrmann CW, et al. Painful lumbar disk derangement: relevance of endplate abnormalities at MR imaging. Radiology. 2001;218:420–7.
31. Waddell G. 1987 Volvo award in clinical sciences: a new clinical model for the treatment of low-back pain. Spine. 1987;12:632–44. https://doi.org/10.1097/00007632-198709000-00002.
32. Kjaer P, Leboeuf-Yde C, Korsholm L, Sorensen JS, Bendix T. Magnetic resonance imaging and low back pain in adults: a diagnostic imaging study of 40-year-old men and women. Spine. 2005;30:1173–80. https://doi.org/10.1097/01.brs.0000162396.97739.76.
33. Kjaer P, Korsholm L, Bendix T, Sorensen JS, Leboeuf-Yde C. Modic changes and their associations with clinical findings. Eur Spine J. 2006;15:1312–9. https://doi.org/10.1007/s00586-006-0185-x.
34. Albert HB, Manniche C. Modic changes following lumbar disc herniation. Eur Spine J. 2007;16:977–82. https://doi.org/10.1007/s00586-007-0336-8.
35. Kuisma M, Karppinen J, Niinimaki J, Ojala R, Haapea M, Heliovaara M, et al. Modic changes in endplates of lumbar vertebral bodies: prevalence and association with low back and sciatic

pain among middle-aged male workers. Spine. 2007;32:1116–22. https://doi.org/10.1097/01.brs.0000261561.12944.ff.
36. Williams FM, Manek NJ, Sambrook PN, Spector TD, Macgregor AJ. Schmorl's nodes: common, highly heritable, and related to lumbar disc disease. Arthritis Rheum. 2007;57:855–60. https://doi.org/10.1002/art.22789.
37. Hamanishi C, Kawabata T, Yosii T, Tanaka S. Schmorl's nodes on magnetic resonance imaging. Their incidence and clinical relevance. Spine (Phila Pa 1976). 1994;19:450–3.
38. Boden SD, Davis DO, Dina TS, Sunner JL, Wiesel SW. Postoperative diskitis: distinguishing early MR imaging findings from normal postoperative disk space changes. Radiology. 1992;184:765–71.
39. Modic MT, Feiglin DH, Piraino DW, Boumphrey F, Weinstein MA, Duchesneau PM, et al. Vertebral osteomyelitis: assessment using MR. Radiology. 1985;157:157–66.
40. Kuisma M, Karppinen J, Niinimaki J, Kurunlahti M, Haapea M, Vanharanta H, et al. A three-year follow-up of lumbar spine endplate (Modic) changes. Spine. 2006;31:1714–8. https://doi.org/10.1097/01.brs.0000224167.18483.14.
41. Toyone T, Takahashi K, Kitahara H, Yamagata M, Murakami M, Moriya H. Vertebral bone-marrow changes in degenerative lumbar disc disease: an MRI study of 74 patients with low back pain. J Bone Joint Surg Br. 1994;76:757–64.
42. Aprill C, Bogduk N. High-intensity zone: a diagnostic sign of painful lumbar disc on magnetic resonance imaging. Br J Radiol. 1992;65:361–9.
43. Peng B, Hou S, Wu W, et al. The pathogenesis and clinical significance of a high-intensity zone (HIZ) of lumbar intervertebral disc on MR imaging in the patient with discogenic low back pain. Eur Spine J. 2006;15:583–7.
44. Khan I, Hargunani R, Saifuddin A. The lumbar high-intensity zone: 20 years on. Clin Radiol. 2014;69:551–8.
45. Wang H, et al. Correlation between High-Intensity Zone on MRI and Discography in Patients with Low Back Pain. Ed. Weisheng Zhang. Medicine. 2017;96(30):e7222. *PMC*. Web. 22 Sept. 2018.
46. Schellhas KP, Pollei SR, Gundry CR, et al. Lumbar disc high-intensity zone. Correlation of magnetic resonance imaging and discography. Spine (Phila Pa 1976). 1996;21:79–86.
47. Saifuddin A, Braithwaite I, White J, et al. The value of lumbar spine magnetic resonance imaging in the demonstration of anular tears. Spine (Phila Pa 1976). 1998;23:453–7.
48. Walsh TR, Weinstein JN, Spratt KF, et al. Lumbar discography in normal subjects. A controlled, prospective study. J Bone Joint Surg Am. 1990;72:1081–8.
49. Wang ZX, Hu YG. High-intensity zone (HIZ) of lumbar intervertebral disc on T2-weighted magnetic resonance images: spatial distribution, and correlation of distribution with low back pain (LBP). Eur Spine J. 2012;21:1311–5.
50. Carragee EJ, Paragioudakis SJ, Khurana S. 2000 Volvo Award winner in clinical studies: lumbar high-intensity zone and discography in subjects without low back problems. Spine (Phila Pa 1976). 2000;25:2987–92.
51. Lee KS, Doh JW, Bae HG, et al. Diagnostic criteria for the clinical syndrome of internal disc disruption: are they reliable? Br J Neurosurg. 2003;17:19–23.
52. Ito M, Incorvaia KM, Yu SF, et al. Predictive signs of discogenic lumbar pain on magnetic resonance imaging with discography correlation. Spine (Phila Pa 1976). 1998;23:1252–8, discussion 1259–1260.
53. Teraguchi M, Yoshimura N, Hashizume H, et al. The association of combination of disc degeneration, end plate signal change, and Schmorl node with low back pain in a large population study: the Wakayama spine study. Spine J. 2015;15:622–8.
54. Ihnatsenka B, Boezaart AP. Ultrasound: basic understanding and learning the language. Int J Shoulder Surg. 2010;4(3):55–62. *PMC*. Web. 22 Sept. 2018.
55. Peetrons P. Ultrasound of muscles. Eur Radiol. 2002;12:35–43, Jan.
56. Lee JC, Healy JC. Normal sonographic anatomy of the wrist and hand. Radiographics. 2005;25:1577–90.
57. Allison SJ, Nazarian LN. Musculoskeletal ultrasound: evaluation of ankle tendons and ligaments. AJR Am J Roentgenol. 2010;194:W514.
58. Morvan G, Busson J, Wybier M, Mathieu P. Ultrasound of the ankle. Eur J Ultrasound. 2001;14:73–82.
59. Peetrons P, Creteur V, Bacq C. Sonography of ankle ligaments. J Clin Ultrasound. 2004;32:491–9.
60. Papatheodorou A, Ellinas P, Takis F, Tsanis A, Maris I, Batakis N. US of the shoulder: rotator cuff and non-rotator cuff disorders. Radiographics. 2006;26:e23.
61. Buchanan BK, DeLuca JP, Lammlein KP. Technical innovation case report: ultrasound-guided prolotherapy injection for insertional achilles calcific tendinosis. Case Rep Orthop. 2016;2016:1560161, 4 pages.
62. Horikawa A, Miyakoshi N, Shimada Y, Kodama H. The relationship between osteoporosis and osteoarthritis of the knee: a report of 2 cases with suspected osteonecrosis. Case Rep Orthoped. 2014;2014:514058, 6 pages.
63. Hasz MW. Diagnostic testing for degenerative disc disease. Adv Orthoped. 2012;2012:413913, 7 pages.
64. Donovan A, Schweitzer M, Bencardino J, Petchprapa C, Cohen J, Ciavarra G. Correlation between rotator cuff tears and systemic atherosclerotic disease. Radiol Res Pract. 2011;2011:128353, 7 pages.
65. Fukuda A, Nishimura A, Nakazora S, Kato K, Sudo A. Entrapment of common peroneal nerve by surgical suture following distal biceps femoris tendon repair. Case Rep Orthoped. 2016;2016:7909805. , 3 pages.
66. Solomou A, Kraniotis P, Rigopoulou A, Petsas T. Frequent benign, nontraumatic, noninflammatory causes of low back pain in adolescents: mri findings. Radiol Res Pract. 2018;2018:7638505, 5 pages.
67. Sugiura K, Tonogai I, Matsuura T, et al. Discoscopic findings of high signal intensity zones on magnetic resonance imaging of lumbar intervertebral discs. Case Rep Orthoped. 2014;2014:245952, 5 pages.