Introduction

COVID-19, the clinical syndrome produced by infection with SARS-CoV-2, can result in multisystem organ dysfunction, including respiratory failure and hypercoagulability, which can lead to critical illness and death. Musculoskeletal (MSK) manifestations of COVID-19 are common but have been relatively underreported, possibly because of the severity of manifestations in other organ systems. Additionally, patients who have undergone sedation and who are critically ill are often unable to alert clinicians of their MSK symptoms. Furthermore, some therapeutic measures such as medications and vaccinations can worsen existing MSK symptoms or cause additional symptoms. Symptoms may persist or occur months after the initial infection, known as post-COVID condition or long COVID. As the global experience with COVID-19 and the vaccination effort increases, certain patterns of MSK disease involving the bones, muscles, peripheral nerves, blood vessels, and joints have emerged, many of which are likely related to a hyper-inflammatory host response, prothrombotic state, or therapeutic efforts rather than direct viral toxicity. Imaging findings for various COVID-19-related MSK pathologic conditions across a variety of modalities are being recognized, which can be helpful for diagnosis, treatment guidance, and follow-up.

The online slide presentation from the RSNA Annual Meeting is available for this article.

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SA-CME LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

- Discuss the pathophysiologic mechanisms associated with the most commonly seen MSK manifestations of COVID-19.
- Describe imaging findings and patterns of disease involving the common COVID-19 MSK manifestations at various modalities.
- Recognize MSK MRI findings following COVID-19 vaccine administration, as well as abnormalities that can be seen in patients with persistent pain after receiving the vaccine.

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COVID-19 reported fatigue, while spine pain was present in 71% of patients and myalgias and arthralgias were noted in 61% and 44% of patients, respectively. As the global experience with COVID-19 increases, it is becoming more widely recognized that MSK symptoms, many of which have described imaging findings, can lead to prolonged disability after recovery from the initial infection (5). Furthermore, COVID-19 vaccinations have resulted in rare MSK-related complications, which have additional imaging findings.

In this article, we review the current understanding of the pathophysiology of COVID-19 and the rare vaccine-related complications, particularly as they relate to MSK manifestations, as well as currently available descriptions of imaging findings in these conditions.

Pathophysiology of SARS-CoV-2 Infection

SARS-CoV-2 is an enveloped single-stranded positive-sense messenger RNA (mRNA) virus from the same family of coronaviruses as severe acute respiratory syndrome coronavirus 1 (SARS-CoV-1) and Middle East respiratory syndrome coronavirus (MERS-CoV) that cause severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS), respectively. SARS-CoV-1 and SARS-CoV-2 have a high degree of genetic and conformational homology and both target the angiotensin-converting enzyme 2 (ACE2) receptor. In the MSK system, skeletal muscle, adipocytes, and endothelial cells in some tissues express ACE2 receptors, representing potential targets for SARS-CoV-2 infection, which may explain some MSK manifestations of disease (6).

Three main pathophysiologic pathways have been proposed to explain the effects of COVID-19 in the MSK system, including the cytokine storm, development of a prothrombotic state, and autoimmunity. The cytokine storm is an inflammatory cascade triggered by the virus that causes elevations in many cytokine levels. Elevated interleukin 6 (IL-6) levels, in particular, have correlated well with more severe disease (7). There is debate in the literature about the term cytokine storm, since the circulating levels of many cytokines may be only modestly elevated despite an inflammatory dysregulation, similar to that in some patients with sepsis. Therefore, some authors have suggested that the term systemic inflammatory response more accurately describes this phenomenon (8).

Platelet hyperreactivity, which could result from direct viral invasion or indirect activation by cytokine release, and activation of the complement system may cause further cytokine release, stimulating the coagulation cascade and suppressing fibrinolysis. Additionally, patients with severe COVID-19 may have limited mobility, which promotes vascular stasis, platelet and erythrocyte aggregation, and thrombosis (9).

Patients with COVID-19 have developed several autoimmune conditions, such as rheumatoid arthritis (RA), psoriatic arthritis, and systemic lupus erythematosus, after the onset of infection (10). Up to 45% of COVID-19 patients exhibit at least one circulating autoantibody. Higher concentrations of autoantibodies often result in more severe disease (7). There is debate in the literature about the term sepsis. Therefore, some authors have suggested that autoimmunity plays a role in the pathogenesis of COVID-19. SARS-CoV-2 has several epitopes that cross-react with host antigens and could result in autoimmune conditions. For example, Lucchese and Flöel (11) showed cross-reactivity of SARS-CoV-2 epitopes with those on heat shock proteins 90 and 60, which could contribute to the development of Guillain-Barré syndrome (GBS). Autoimmunity due to SARS-CoV-2 infection may also be related to superantigen reactivity (12).

Although the specific mechanisms resulting in clinical symptoms in COVID-19 are being assessed, a few common pathways, including systemic inflammatory dysregulation, autoimmunity, and hypercoagulability, often occur together to produce end-organ damage in a variety of tissues (Fig 1).

COVID-19 in MSK System

Within the MSK system, COVID-19 may affect bones, muscles, the coagulation pathway, periph-
lent of 200 mg methylprednisone) for 1 week or more (15). Other risk factors include male sex, use of multiple types of corticosteroids, alcohol or tobacco use, and cardiovascular and/or cerebrovascular disease. The most common sites of ON in patients with SARS and COVID-19 are the femoral head, knee, humeral head, talus, and calcaneus. Although the precise cause of steroid-induced ON is unknown and probably multifactorial, it likely involves osteoblastic suppression and osteocyte apoptosis, leading to bone resorption, along with the development of microthrombi that result in decreased perfusion and intraosseous adipogenesis. This increases intramedullary pressure and may cause vascular occlusion and venous stasis (13). Agarwala et al (16) reported the development of ON in COVID-19 patients with a lower steroid dose and over a shorter time compared with patients without COVID-19. There are reports that symptoms of hemoglobinopathies, such as acute chest syndrome in sickle cell disease, may worsen after COVID-19 infection, and some patients may develop ON. Sheha et al (17) reported a case of a patient who was discovered to have sickle cell trait after developing ON during COVID-19 infection.

The imaging findings of periarticular ON in COVID-19 patients are similar to those seen in patients with other conditions. Many severely ill ner nerves, and joints. Additionally, patients with long COVID or those who have been treated or vaccinated can present with MSK symptoms. While the causes of these conditions are complex and likely multifactorial, some themes have emerged as our understanding evolves.

**Osseous Manifestations**

COVID-19 patients may present with articular or periarticular pain, which can be secondary to osteonecrosis (ON). Cortical bone is a potential site of direct infection by SARS-CoV-2 owing to its expression of ACE2 receptors (13). ON is a well-described condition associated with the original SARS, reported in 5%–58% of patients. Proposed mechanisms include corticosteroid administration, hypercoagulability, vascular inflammation (including endothelial inflammation and perivascular leukocyte aggregation), and bone resorption by stimulation of ACE2 receptors (14).

Corticosteroid therapy, likely the leading cause of ON, is administered to severely and critically ill patients who require ventilation to reduce morbidity and mortality and may be used in high doses over prolonged periods. The risk of ON was greatly increased in those patients with SARS who received higher doses (cumulative dose greater than or equal to the equivalent of 4000 mg methylprednisone and peak dose the equivalent of 200 mg methylprednisone) for 1 week or more (15). Other risk factors include male sex, use of multiple types of corticosteroids, alcohol or tobacco use, and cardiovascular and/or cerebrovascular disease. The most common sites of ON in patients with SARS and COVID-19 are the femoral head, knee, humeral head, talus, and calcaneus. Although the precise cause of steroid-induced ON is unknown and probably multifactorial, it likely involves osteoblastic suppression and osteocyte apoptosis, leading to bone resorption, along with the development of microthrombi that result in decreased perfusion and intraosseous adipogenesis. This increases intramedullary pressure and may cause vascular occlusion and venous stasis (13). Agarwala et al (16) reported the development of ON in COVID-19 patients with a lower steroid dose and over a shorter time compared with patients without COVID-19.

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COVID-19 patients undergo periodic chest CT and abdominal CT examinations for respiratory and gastrointestinal symptoms, which may help detect radiographically occult ON of the humeral and femoral heads (17) (Fig 2). MRI remains the most sensitive modality for detecting occult ON with stage-related characteristic imaging findings (16).

COVID toes, also known as chilblain-like or pernio-like lesions, are localized acral cutaneous erythematous eruptions commonly involving the toes more often than the fingers. This is most commonly seen in teens and young adults, especially in immunocompetent patients or those with mild COVID-19 symptoms. Its cause is unknown. However, several authors have suggested vascular and immunologic causes, including vasospasm, perivascular lymphocytic aggregation involving small dermal vessels, endothelial damage, and fibrin microthrombi (18). These lesions are usually evident at visual inspection without the need for imaging. However, COVID toes may be associated with ON of the small bones of the feet, diagnosed at MRI, in patients without other common causes for ON (Fig 3).

There are several case reports of heterotopic ossification (HO) in critically ill COVID-19 patients, which may cause joint pain. Stoira et al (19) reported a prevalence of 19.2% (10 of 52 patients) in the largest case series to date. According to current reports, the hips and shoulders have been most commonly involved, followed by the elbows, similar to patients who develop HO secondary to other conditions (19). Prolonged immobilization, especially in patients who have required ventilation, is the biggest factor contributing to the development of HO. However, systemic inflammation seen in COVID-19 patients may upregulate the IL-6/tumor necrosis factor α (TNF-α) pathway and stimulate mesenchymal cells to release prostanoid E2, which have been associated with HO development (20).

The early imaging findings of HO are better depicted at CT than radiography, with peripheral soft-tissue mineralization gradually maturing to cortical bone with internal cancellous bone,
which subsequently may attach to the underlying bone surface (20) (Fig 4). Initial MRI findings may be confusing with avidly enhancing extensive soft-tissue edema and distortion. As HO matures, the peripheral low-signal-intensity rim of calcification confirms the diagnosis (21).

**Muscle Manifestations**

COVID-19 may manifest with myalgias and fatigue unrelated to disease severity. Severe muscle injury can be associated with multisystem organ failure. This includes myoglobinuria in the setting of rhabdomyolysis, which can lead to renal insufficiency, and involvement of respiratory muscles, which can worsen dyspnea related to respiratory failure (22,23). These complications are more common in patients who are older and critically ill. The cause is likely multifactorial, including immune-mediated damage, intensive care unit–acquired weakness, toxic myopathy, and possibly direct viral damage, with certain factors playing a larger role in the development of muscle injury in particular patients. COVID-19–related muscle pathologic conditions can be divided into four categories—myalgias, myositis, myonecrosis, and rhabdomyolysis—which can help imaging assessment. Noninflammatory myalgias are generally self-limited and treated conservatively. If a patient with myalgias undergoes imaging with CT or MRI, the muscle should look normal, without fascial thickening, fluid, or enhancement (24).

Myositis, a true inflammation of skeletal muscle, can occur in a variety of conditions, including SARS-CoV-2 infection. Serum muscle-specific markers can be transiently elevated. MR images may show areas of skeletal muscle and fascial and subcutaneous edema with hyperintense signal with fluid-sensitive sequences and postcontrast enhancement (25) (Fig 5). Although CT images may also show muscle thickening and effacement of intramuscular fat and fascial planes, MR images can better depict muscle edema, fascial stranding, and postcontrast enhancement.

Myonecrosis has several causes, including radiation therapy, diabetes mellitus, immobilization, exercise, medications (eg, statins), and therapeutic ablation (25). There may be mild transient elevation of serum creatine kinase levels. However, urine myoglobin levels are normal. Myonecrosis can result in substantial pain, and patients are treated supportively. MRI and CT show similar findings, although MRI is more sensitive for helping detect areas of myonecrosis. Two MRI patterns have been described: type 1 myonecrosis reflects potentially reversible muscle ischemia, and type 2 myonecrosis results from irreversible muscle infarction. In type 1 myonecrosis, the MRI findings are similar to those of myositis, with areas of diffuse muscle and fascial edema with fluid-sensitive sequences and postcontrast enhancement. In type 2 changes, there are nonenhancing intramuscular areas of variable sizes, often with thin peripheral enhancement (Fig 6). Frequently observed is a stipple sign, with linear or curvilinear foci of enhancement within the area of necrosis on long-axis images that have a dotlike appearance in cross-section planes, possibly representing residual viable myofibers or inflamed crossing vessels (26).

Rhabdomyolysis represents significant muscle injury and possibly myonecrosis leading to more pronounced, sustained elevations of serum creatine kinase levels and myoglobinuria, which can result in acute renal injury. In COVID-19 patients, rhabdomyolysis is often symmetric with a proximal lower limb predominance, leading to profound weakness (23). The diagnosis is made clinically, and imaging may help assess the location
and degree of muscle injury. The MRI findings are similar to those of myositis, although there may be areas of nonenhancing type 2 myonecrosis (26).

**Joint Manifestations**

Patients with COVID-19 often present with arthralgias. When there is an identifiable cause, synovitis related to a primary postviral inflammatory arthritis or other rheumatologic conditions such as RA, systemic lupus erythematosus, or psoriatic arthritis may occur in addition to ON and HO as previously mentioned (10).

Post–COVID-19 inflammatory arthritis is an aseptic inflammatory arthropathy likely related to the host immune response. Many patients predictably present 1–3 weeks after the onset of infection with symptoms extending into the recovery phase. Synovitis and clinical symptoms are often responsive to treatments used in other inflammatory arthropathies. This condition is typically monoar-
ticular or oligoarticular and more commonly seen in the lower extremities and in men. The findings may be a manifestation of reactive arthritis, which is also usually monoarticular or oligoarticular (27). In comparison, inflammatory arthritis occurring with other viral infections usually manifests during the acute viremic phase of infection and is more commonly polyarticular (28).

Several possible mechanisms for the development of post–COVID-19 inflammatory arthritis have been proposed, the most common of which is molecular mimicry owing to viral epitopes on the spike protein causing production of autoantibodies. Other mechanisms, such as bystander activation, an antigen-independent stimulation of T cells and/or B cells in sites of inflammation, can also contribute to and exacerbate the autoimmune effects (29). Direct viral injury is considered unlikely as viremia is seen in only about 15% of COVID-19 patients and no detectable viral particles have been recovered in polymerase chain reaction (PCR) testing of arthrocentesis samples of a few patients (10,30).

Although most reported cases of COVID-19–related inflammatory arthropathy represent reactive postviral arthritis, other inflammatory arthropathies such as polyarticular RA, systemic lupus erythematosus, and seronegative spondyloarthropathies have been reported. Cases of seropositive RA have usually been described several weeks after the onset of moderate to severe pulmonary symptoms (10). Some patients present with symptoms of active sacroiliitis, possibly associated with activation or exacerbation of seronegative spondyloarthropathies such as psoriatic arthritis, or post–COVID-19 reactive sacroiliitis. Clinical and imaging features established by the Assessment of SpondyloArthritis International Society (ASAS) can help confirm the diagnosis of seronegative spondyloarthropathy (31). It is unclear if there is an association of human leukocyte antigen B27 (HLA-B27) with seronegative spondyloarthropathies that follow COVID-19 (32).

The imaging findings of post–COVID-19 inflammatory arthritis are uncommonly reported but are similar to those of viral arthritis from other infections. MRI demonstrates monoarticular or oligoarticular synovial thickening and edema of the affected joints, and there may be tenosynovitis, with variable degrees of contrast enhancement (33) (Fig 7). Periarticular erosions are rare. This could be related to early stages of disease or because medications used to treat other manifestations of COVID-19, such as corticosteroids or hydroxychloroquine, may blunt the severity of the synovitis (10). The presence of erosions may suggest an underlying rheumatologic disease such as RA (34). Similarly, US can help detect synovial thickening, which may be heterogeneous or hypoechoic relative to other tissues. There may be variable periarticular vascularity at Doppler US, ranging from no detectable hyperemia, indicating bland synovial proliferation, to extensive hyperemia, consistent
with active synovitis (33). Newer microvascular Doppler US techniques may be more sensitive in detecting active synovitis (35) (Fig 8).

MRI is the most sensitive imaging test for detecting sacroilitis, including symmetric subchondral marrow edema and erosions, particularly in the lower and posterior thirds of the joint (Fig 9). Some patients may demonstrate synovial and capsular edema at fluid-sensitive imaging and synovial and capsular enhancement after intravenous contrast material administration (32). Although chronic changes of sacroilitis described with other conditions, such as subchondral sclerosis, subchondral fat deposition, and ankylosis, have not been described, this may be because longitudinal assessment of patients with COVID-19–related sacroilitis has not been performed.

Ascani et al (36) reported a case series of 12 patients who developed adhesive capsulitis following asymptomatic or mild COVID-19 with symptom occurrence 1.5–3 months following COVID-19 diagnosis. The authors speculated that there was a combination of direct effects from viral infection of fibroblasts and monocytes within the synovium, cytokine dysregulation inducing inflammation and subsequent fibrosis, and lifestyle changes, including decreased physical activity, which contribute to its development (36) (Fig 10).

**Hematologic Manifestations**

COVID-19 is a well-known cause of hypercoagulability. The cause is likely multifactorial and represents the interplay of inflammatory dysregulation, endothelial dysfunction, and venous stasis, often occurring in patients who are immobile and hospitalized. Thromboembolism may involve numerous organ systems. In addition to small vessel thrombosis, which is one of the hallmarks in autopsies of COVID-19 patients with respiratory and multisystem organ failure, large vessel venous thromboembolism is also common (9). Deep venous thrombosis (DVT) occurs in about 20%–25% of hospitalized patients and may result in pulmonary embolism (37). The reported cases of recurrent venous thromboembolism with COVID-19 provide additional evidence of its prothrombotic state.

Imaging is used to confirm the diagnosis, location, and extent of macrothrombosis. Most cases of COVID-19–related DVT have been described in the lower extremities, either in the calf musculature and popliteal vein or the femoral vein with central propagation. Classic findings of acute venous thrombosis are best diagnosed at gray-scale and color and spectral Doppler US (38).

Arterial thrombosis is described in 1%–2% of COVID-19 patients who are critically ill. Although a causal link is unclear, several centers have observed a significantly higher frequency of arterial thrombosis in COVID-19 patients without preexisting atherosclerotic disease or other causes of hypercoagulability, often with multivessel involvement. Critical illness, obesity, male sex, and history of coronary artery disease are the greatest risk factors for developing this complication (39).

CT angiography is the study of choice to help diagnose arterial thrombosis, with classic imaging findings well described in the literature (38). Simultaneous imaging of the contralateral extremity may help to detect subtle abnormalities (Fig 11). MRI may also help provide the diagnosis in patients unable to receive iodinated contrast material. Classic MRI findings include loss of the normal intravascular flow void, arterial distention, perivascular edema, and soft-tissue edema. MR angiography, with a lack of normal enhancement in the area of thrombosis, may contribute to the diagnosis (40).

COVID-19–related coagulopathy resulting in spontaneous MSK hemorrhage is much less common than thromboembolism. Spontaneous bleeding most commonly occurs in skeletal muscle, especially the iliopsoas, as well as the pectoralis major and rectus abdominis muscles (41,42). A recent study showed that critically ill COVID-19 patients were twice as likely to develop a spontaneous iliopsoas hematoma compared with those in the intensive care unit for other reasons (41). Patients may have acute progressive pain, swelling, development of a mass, and possibly skin bruising, with decreasing hemoglobin levels. Larger hematomas may cause mass effect on adjacent structures, such as peripheral nerves, and result in compressive neuropathy (43). Anticoagulation therapy is the biggest risk factor for developing spontaneous muscle hematoma. Other risk factors include older age, obesity, hypertension, and diabetes mellitus (42). Several proposed mechanisms may contribute to the pathogenesis of this phe-
nomenon, including prophylactic anticoagulation therapy and renal damage–associated decreased clearance of anticoagulants. Additionally, SARS-CoV-2 infection and the subsequent cytokine storm may result in endothelial inflammation and damage leading to capillary microtrauma and arterial vulnerability (42).

If the clinical diagnosis is unclear, CT is an excellent initial modality since it can be performed rapidly over large fields of view. Noncontrast imaging will show muscle enlargement with a hyperattenuating collection if the patient is not anemic, possibly with a fluid-hematocrit level. Intravenous contrast material is usually unnecessary. MRI may be performed in patients in whom alternate diagnoses are suspected and can be helpful to assess for compression on surrounding structures. The signal intensities of MSK hematomas are usually heterogeneous depending on the composition of blood products, without internal enhancement in the absence of an underlying mass (41). Compression of motor nerves may result in denervation muscle edema at fluid-sensitive imaging and possibly atrophy at non–fat-suppressed T1-weighted imaging in cases of longer-standing compression (Fig 12). US examination often shows a complex heterogeneous lesion, sometimes with dependent fluid and/or hematocrit levels and septa and without nodular or internal vascularity to indicate an underlying solid mass (44).

Figure 9. Sacroiliitis in a 55-year-old man with a history of COVID-19 2 months earlier and subsequent low back and perianal pain. A full workup failed to show a gastrointestinal cause for the patient’s symptoms. Given the patient’s age (>45 years) and the lack of classic inflammatory back pain symptoms or history of psoriasis or inflammatory bowel disease, an axial spondyloarthritis was felt to be unlikely, and the HLA-B27 receptor status is unknown. (A) Coronal oblique T1-weighted non–fat-suppressed MR image of the sacroiliac joints shows bilateral sacroiliac erosions with irregularity of the subchondral bone plates (arrows) that are more pronounced on the iliac side of the joints. (B, C) Coronal oblique T2-weighted fat-suppressed (B) and coronal oblique T1-weighted fat-suppressed postcontrast (C) MR images of the same area show bone marrow edema and enhancement in the areas of erosion (arrows), suggestive of active lesions. The findings are slightly worse on the right, where they extend more cranially and involve the sacral side of the joint to a greater degree.

Figure 10. Adhesive capsulitis in a 46-year-old woman who developed atraumatic left shoulder pain and decreased range of motion 2 months after recovering from mild symptoms of COVID-19. (A) Coronal T2-weighted fat-suppressed MR image of the shoulder shows typical findings often seen with adhesive capsulitis, including an edematous thickened inferior glenohumeral ligament (IGHL), particularly at its humeral attachment (arrows). (B) Sagittal T2-weighted fat-suppressed MR image of the shoulder again shows edema of the IGHL (solid arrows), with additional mild rotator interval edema and thickening of the coracohumeral ligament (dashed arrow). Mild supraspinatus myotendinous junction edema is also seen (arrowhead). (C) Sagittal T1-weighted non–fat-suppressed MR image of the same area confirms thickening and ill definition of the coracohumeral ligament (dashed arrow). The fat within the rotator interval is still partially preserved. The IGHL also appears thickened diffusely (solid arrows). The MRI findings are compatible with the freezing or inflamed phase of adhesive capsulitis.
Peripheral Nerve Manifestations

COVID-19–associated peripheral neuropathies are more commonly described in patients who are critically ill and require ventilation and have several possible causes, including GBS, toxic neuropathy, critical illness polyneuropathy (CIP), compressive neuropathy, and position-related neuropathy (51). While damage from direct viral infection was initially considered a possible mechanism, more recent evidence suggests other causes are likely, since viral particles are rarely recovered in PCR testing of the nerves and cerebrospinal fluid in most patients.

Secondary Infections

Secondary infections, such as bacteremia and urinary or respiratory tract infections, are common and may be seen in up to 50% of hospitalized COVID-19 patients (45). Otherwise, spontaneous MSK infections, such as intramuscular abscesses and septic arthritis, are uncommon in immunocompetent patients. One orthopedic study showed an unusually high frequency of periprosthetic infections, soft-tissue abscesses, and septic arthritis over 1 year in 12 of 90 hospitalized COVID-19 patients (46). Talamonti et al (47) described six hospitalized COVID-19 patients with spinal epidural abscesses within a 3-month span, most without risk factors for developing MSK infections. Risk factors such as diabetes mellitus may contribute to increased infection rate. *Staphylococcus aureus* is the most common causative organism, although unusual organisms like *Fusobacterium* and *Actinomyces* species have also been described.

While the causes of these infections are unknown, COVID-19 patients frequently develop lymphopenia, which could lead to a consumptive immunocompromise. Alternatively, vascular endothelial damage may allow virulent organisms to more easily penetrate vessels walls and seed surrounding tissues. Direct viral damage seems unlikely since a few case reports in which abscess fluid was recovered failed to show viral particles at PCR testing (48). Imaging of soft-tissue abscesses, including with US, CT, and MRI, is well described in the literature. MRI is the best modality to assess axial and appendicular MSK infections including spinal epidural abscesses (Fig 13). Intravenous contrast material is helpful for detecting rim-enhancing abscesses and may be substituted with diffusion-weighted sequences as needed (49,50).

**Figure 11.** Arterial and venous lower extremity thrombosis in a 49-year-old man with a history of COVID-19 who presented with left lower extremity pain, swelling, and pulselessness. (A) Axial CT angiographic image of the lower extremities at the level of the femoral condyles shows contrast material opacifying the right popliteal artery and vein (solid arrow) but not the left (dashed arrow), consistent with thrombosis. (B) More distal axial CT angiographic image of the calves shows near-complete nonopacification of the right popliteal vein with a thin rim of surrounding contrast material (solid arrow), indicating near-occlusive thrombosis. In the left lower leg, there is subcutaneous stranding (arrowhead), consistent with soft-tissue edema. The multifocal arterial and venous thrombi are suggestive of a hypercoagulable state. (C) Three-dimensional reformatted coronal CT angiographic image shows absence of opacification of the left popliteal artery and its major branches (dashed arrow) owing to the arterial thrombosis. In comparison, the right popliteal artery and its major branches are well opacified (solid arrow), consistent with patency.
Risk factors include male sex, age greater than 50 years, diabetes mellitus, hypertension, and prolonged intensive care unit stay. In a mini-review by Finsterer et al, the most commonly reported COVID-19–related peripheral neuropathy was GBS. Whether GBS is truly a consequence of COVID-19 has been debated, although several centers have reported a significant increase in GBS cases during the COVID-19 pandemic in patients without other recognized causes. Although GBS in COVID-19 can be parainfectious, most reported cases are postinfectious, starting about 5–10 days after the onset of COVID-19 symptoms.

MRI is the best imaging modality to help assess GBS and is usually performed to exclude other possible causes, such as central nervous system insults. Lumbar spine MRI may show distal spinal cord hyperintensity at T2-weighted imaging and nerve root thickening of the cauda equina, with surface enhancement of these nerve roots as well as the conus medullaris (Fig 14). Occasionally, there can be leptomeningeal and spinal nerve root enhancement on brain MR images.

CIP occurs in critically ill patients and results in a symmetric lower limb–predominant flaccid muscle weakness that can involve the phrenic nerve and muscles of respiration, similar to GBS.
Unlike GBS, it rarely involves the facial muscles. Several factors can contribute to its pathogenesis. Imaging is nonspecific and usually performed to exclude other causes. MRI can show muscle edema and possibly fatty infiltration, worse in the lower extremities, and can help to direct muscle biopsy when indicated (5).

Other categories of peripheral neuropathy with described imaging findings in COVID-19 patients include position-related, compressive, and postinfectious neuropathies that may be related to thrombotic microangiopathy (Fig 15). Prolonged prone positioning can cause excessive traction and ischemia of peripheral nerves. Unlike GBS and CIP, this is more frequent in younger patients, commonly involves the upper extremities, particularly the ulnar nerve and cords of the brachial plexus, and may be unilateral or asymmetric. Lower extremity neuropathies most often involve the sciatic and common peroneal nerves (57). There may be localized nerve damage from microvascular injury, cytokine-mediated inflammation, and possibly compression within fibro-osseous tunnels such as the carpal and cubital tunnels (58).

Both high-spatial-resolution MR neurography and US are useful in characterizing peripheral neuropathies. Intravenous contrast material is usually unnecessary since peripheral nerves in these conditions do not generally enhance. However, contrast material can help suppress vascular signal intensity, especially during isotropic three-dimensional short $\tau$ inversion-recovery (STIR) techniques, making the distinction between peripheral nerves and blood vessels clearer. There may also be associated muscle edema with fluid-sensitive sequences, and/or decreased muscle bulk and muscle fatty atrophy on T1-weighted non–fat-suppressed images corresponding to a peripheral nerve distribution (51) (Fig 16).

US is an excellent modality for assessing peripheral nerves. Similar to MRI, the nerves may appear thickened in areas of neuropathy, and one or more fascicles may be enlarged and hypoechoic. Alternatively, there may be effacement of the normal fascicles, producing a swollen featureless appearance (59). Newer microvascular Doppler US techniques may detect slow-flow intraneural vascularity indicating focal nerve abnormality (60).

Several medications specifically used in treating COVID-19, such as hydroxychloroquine and remdesivir, can result in toxic neuropathies, which are usually diagnosed clinically and aided by electrodiagnostic examinations rather than imaging examinations (51).

Figure 14. GBS in a 15-year-old adolescent girl with COVID-19 with progressive ascending lower extremity pain and weakness. The patient was diagnosed with COVID-19–related acute motor axonal neuropathy (AMAN), a variant of GBS, and myopathy due to either AMAN, critical illness myopathy, or both. (A, B) Axial T1-weighted non–fat-suppressed postcontrast MR image of the lumbar spine at the L3 level (A) and sagittal T1-weighted fat-suppressed postcontrast MR image of the lumbar spine through the left neural foramina (B) show thickening and enhancement of the nerve roots (arrows). (C) Axial T1-weighted non–fat-suppressed MR image of the mid thighs shows diffuse left thigh muscle atrophy, particularly in the posterior compartment, with more subtle right thigh posterior compartment decreased muscle bulk. (D) Axial short $\tau$ inversion-recovery (STIR) MR image at the same level shows mild diffuse left thigh muscle edema and subtle left thigh anterior compartment muscle edema, which may indicate areas of greater disease activity.
Post–COVID-19 Condition

Post–COVID-19 condition may be seen in 10%–20% of patients, generally in younger individuals between 20 and 50 years of age. In October 2021, the World Health Organization released the following consensus definition: “Post COVID-19 condition occurs in individuals with a history of probable or confirmed SARS CoV-2 infection, usually 3 months from the onset of COVID-19 with symptoms and that last for at least 2 months and cannot be explained by an alternative diagnosis…. Symptoms may be new onset following initial recovery from an acute COVID-19 episode or persist from the initial illness.” Several of these symptoms, such as fatigue, shortness of breath, arthralgias, muscle pain and/or spasms, and postexertional malaise, can be related to the chronic impact of COVID-19 on skeletal muscle.

Long-standing COVID-19–related myopathy can result in muscle weakness, spasms, and exercise intolerance and is associated with decreased muscle bulk and fatty infiltration. These may be associated with denervation atrophy, deconditioning and sarcopenia, immune-mediated myopathy, toxic myopathy, and nutritional deficiencies. The findings may be unilateral or asymmetric depending on the involved nerves.

MRI, CT, or US can be performed to detect well-described chronic muscle changes. If there is enough fatty infiltration, the overall muscle cross-sectional area may not be significantly reduced. In these cases, fat quantification techniques, such as with Dixon MRI, can provide a measure of muscle atrophy that can aid serial assessment. Intravenous contrast material is generally unnecessary, since denervated atrophic muscle usually does not enhance.

Although US is helpful in detecting focal or patchy muscle atrophy compared with normal-appearing adjacent muscle, it may be more difficult to detect global balanced muscle abnormalities. Shear wave sonoelastography can be useful to quantitatively assess muscle stiffness and is an emerging technique that can help detect early abnormalities and follow progression of atrophy.

Sarcopenia, which is generally seen with aging and deconditioning, can be more pronounced in COVID-19 patients who are critically ill and may affect the muscles of respiration, inhibiting the ability to wean patients from ventilators. Although this is likely indirectly due to COVID-19 through prolonged immobilization, diffuse COVID-19–related myopathy may also contribute to its development. CT and MRI are commonly performed to detect and follow findings of sarcopenia. Assessment of skeletal muscle cross-sectional area, normalized with respect to patient sex, age, and occasionally body mass index, is commonly performed at the L3 vertebral level, which is the imaging reference standard. Recent work has validated that cross-sectional area measurements between T10 and T12 or at the T4 level can be useful surrogates in chest CT examinations performed in many patients with COVID-19.

Respiratory muscle dysfunction can involve the diaphragm. Long-standing diaphragmatic dysfunction is seen in up to 76% of COVID-19 patients and can contribute to dyspnea in the post–COVID-19 condition. Several factors likely contribute to its pathogenesis, including sarcopenia, critical illness myopathy, and ventilator-induced dysfunction. Diaphragmatic dysfunction historically has been assessed fluoroscopically by using the sniff test but can be assessed over successive breaths at M-mode US. With quiet breathing, the normal excursion has been reported as 10 mm for men and 9 mm for women. In cases that are equivocal during quiet breathing, assessing excursion during deep breathing (with deep inspiration and terminal expiration) may be useful. Normal values for excursion with deep breathing are 47...
The value of diaphragmatic US in patients who require ventilators is still being assessed and may also depend on the mode of ventilation.

Vaccine-related Manifestations

Since December 2020, COVID-19 vaccinations have been used to decrease the severity of disease and the likelihood of disease transmission. To date, more than 11.6 billion doses of various COVID-19 vaccines in 5.1 billion people have been administered worldwide, which represents 67% of the world’s population (1). While COVID-19 vaccination is considered very safe and effective, several reactions similar to those of other vaccines have been reported, including localized pain and swelling at the injection site and possibly myalgias and arthralgias for a few days following the injection. Rarely, MSK symptoms can persist for months and may even become chronic.

Most adult vaccines are administered intramuscularly into the lateral or posterolateral deltoid muscle, about 3 cm inferior to the lateral margin of the acromion. Postvaccination shoulder pain lasting more than a few days and not well controlled by common conservative measures occurs in up to 2% of injections (69). The most common postvaccination condition is shoulder injury related to vaccine administration (SIRVA), which was first described in 2006 (70). Classically, patients develop rapid onset of shoulder pain and decreased range of motion within 48 hours of vaccination. This is most often following the influenza vaccination, probably owing to the frequency that this vaccine is administered. SIRVA is likely related to a localized immunologic reaction to the injectate, and several authors have suggested that
this is more common when there has been a prior exposure that sensitizes a patient. SIRVA usually occurs with improper positioning of the needle tip, particularly within the upper third of the deltoid, possibly extending into the subacromial-subdeltoid bursa, rotator cuff tendons, glenohumeral joint, or even the humerus, which may result in bursitis, rotator cuff tendinopathy, glenohumeral joint synovitis, adhesive capsulitis, and osteitis and/or osteomyelitis (71) (Fig 20).
Patients with SIRVA related to COVID-19 vaccination seem to differ slightly from those following other vaccinations. First, several patients have developed symptoms 1–2 weeks after injection, and one patient reportedly developed symptoms 8 weeks after the injection (72). In these cases, the presenting symptoms were typical of SIRVA and no other inciting cause was found. Secondly, several reported cases developed after only one dose of the vaccine. These cases suggest that the inflammatory reaction may take some time to develop if there has not been prior exposure. In particular, the Pfizer-BioNTech and Moderna mRNA vaccines appear highly immunogenic and may produce a strong reaction when injected into nontarget synovial tissues (72).

Other injuries may occur if the needle placement is outside of the safe zone within the deltoid muscle. If the needle is too lateral, it may result in axillary nerve injury; if it is too inferior, it can injure the radial neurovascular bundle; if it is too long it could result in chemical osteitis/osteomyelitis of the humerus; and if it is too short, it can result in local skin reaction and subcutaneous fat necrosis (73).

Clustered enlarged axillary lymph nodes, commonly seen following COVID-19 vaccination, are likely reactive and may last for several months (74). Localized muscle edema with overlying mild subcutaneous and fascial edema may be present for a few days or weeks following injection. However, persistent muscle edema with localized shoulder pain may reflect focal myositis (75).

Rotator cuff tendon tears reported in some patients may be preexisting, but the inflammatory response may contribute to symptom development. Periarticular and articular infections are rare, with a single case of glenohumeral septic arthritis and subacromial-subdeltoid bursitis reported (49,76). As with other causes, potential COVID-19 vaccine–related rotator cuff, bursal, and other periarticular soft-tissue pathologic conditions may be diagnosed with MRI or US, while MRI remains the primary modality for glenohumeral joint evaluation (77).

There is literature reporting rare inflammatory MSK manifestations following COVID-19 vaccination, including reactive inflammatory arthropathies as well as flares of existing rheumatologic conditions and GBS, suggesting an autoimmune response. In a series of 66 patients reported by Ursini et al (78), the most common rheumatologic condition was a transient symmetric polymyalgia rheumatica-like inflammation (78). Uncommon mild flares of existing rheumatologic conditions such as RA or psoriatic arthritis have been described (79). Imaging studies of postvaccine immune-mediated MSK manifestations have not been well reported.

A meta-analysis showed that GBS following COVID-19 vaccine administration tended to manifest 1–3 weeks after administration of the first dose of the vaccine, more commonly in men and those over 50 years of age. Additionally, patients most commonly suffered from demyelinating polyneuropathy and bifacial weakness,
which may be defining characteristics of post–COVID-19 vaccine-related GBS (80).

Conclusion
COVID-19, a clinical syndrome produced by SARS-CoV-2 infection, has wide-reaching effects throughout the body, including the MSK system. The impact of these MSK manifestations on long-term patient outcomes has yet to be determined. Rare COVID-19 vaccine–related complications have also been reported. It is important for radiologists to understand the common patterns of COVID-19 involvement in the MSK system, including relevant imaging findings seen with various modalities, as imaging can help identify several MSK manifestations of COVID-19, assess the distribution of disease, and provide quantifiable measurements to follow disease progression.

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