Introduction

The coronavirus (CoV) are members of the order Nidovirales, family Coronaviridae, and genus Coronavirus. The official name of this virus is SARS-CoV-2. Seven CoV species are known to infect people. Only MERS-CoV and SARS-CoV have the capacity to lead to serious human illness.

The remaining are linked to minor respiratory illnesses like the common cold. The average number of days that COVID takes to incubate is two to seven (range of one to ten). The single-stranded (positive-sense) RNA virus, SARS-CoV-2, which causes COVID-19, is known to produce fever, dry cough, fatigue, headache, and loss of taste and smell. Some COVID-19-affected individuals may develop a severe form of pneumonia, eventually leading to acute respiratory distress syndrome (ARDS) and respiratory failure. Recently published case reports have emphasized additional uncommon and unusual presentations of infection in individuals affected by the SARS-CoV-2 virus. SARS-CoV has the necessary potential to cause community and nosocomial transmission and result in severe morbidity and mortality.

The virus also seems to involve the musculoskeletal system. Though these are believed to be infrequent presentations of infection in individuals affected by the SARS-CoV-2 virus. SARS-CoV has the necessary potential to cause community and nosocomial transmission and result in severe morbidity and mortality.

Case Presentation

Presentation and examination

A 47-year-old male patient had a history of positive reverse transcription-polymerase chain reaction (RT-PCR) test for SARS-CoV-2 infection three months before, for which he was hospitalized and subsequently underwent treatment in the form of antibiotics, prophylactic thrombolytics, and supportive therapy. At the time of presentation, he came to our tertiary care center in central India with complaints of pain and swelling over the right lower limb for 20 days. The pain was insidious in onset, gradual in progression, and was initially present over the posterior aspect of the right lower limb at the calf region and later progressed to the anterior compartment. The pain aggravated during physical activities and relieved on rest. The swelling was present over the right lower limb, which was smooth, diffuse in nature, and associated with redness and local rise of temperature on the overlying skin. There was no restriction to movements of the joint above and below the swelling, and no associated complaints of discharge from the swelling or visible pulsations were noted. The patient had no previous history of trauma, endocrine disorders, similar complaints in the family, blood disorders, or genetic disorders. The patient had no history of usage of illegal-lowering drugs, alcohol, colchicine, glucocorticoids, or antimarial drugs in the past. The patient was initially diagnosed with a urinary tract infection and was treated accordingly.

Laboratory investigations

The values of the complete blood count were as follows: Hemoglobin 14.5 g/dL, hematocrit 44%, RBC count 4.9 x 10^12/microL, mean corpuscular volume (MCV) 89 fL, mean corpuscular haemoglobin concentration (MCHC) 35, red blood cell distribution width (RDW) 12%, platelet count 230 x 10^3/microL, WBC count 15,000/microL. The WBC count was found to be raised. Biochemical tests for detection of inflammatory markers were performed, which revealed C-reactive protein (CRP) 30mg/L, erythrocyte sedimentation rate (ESR) 15,000/microL, mean corpuscular volume (MCV) 89 fL, mean corpuscular hemoglobin concentration (MCHC) 35, red blood cell distribution width (RDW) 12%, platelet count 230 x 10^3/microL, WBC count 15,000/microL. The CRP value of 30 mg/L was significantly high. Biochemical tests for detection of inflammatory markers were performed, which revealed C-reactive protein (CRP) 30mg/L, erythrocyte sedimentation rate (ESR) 48mm/hr, lactate dehydrogenase (LDH) 748IU/L, and creatine kinase (CK) 644IU/L. A significant high value of CRP, ESR, lactate dehydrogenase (LDH), and CK was noted. Differential diagnosis of CoV-19 neuromuscular disease such as encephalitis, myelitis, or Guillain-Barré syndrome were ruled out due to normal cognitive and cerebellar functions with normal reflexes. Tests for infections such as HIV, Hepatitis B and C were negative.

Imaging findings

It made ultrafast MR imaging examination of the swelling showed increased diffuse homogeneous enhancement in the soleus, plantaris, and gastrocnemius muscles in the posterior compartment of the right lower limb. The extensive dystrophic changes and perivascular changes were shown to exhibit enhanced echogenicity that was diffusely distributed along the muscles in the anterior compartment and lateral compartment, respectively. The patient was therefore subjected to undergo magnetic resonance imaging (MRI) for further evaluation. In the MRI of the right lower limb, there was evidence of extensive T2-FLAIR hyperintensities in muscles of the anterior compartment (extensor digitorum longus), lateral (peroneus longus), superficial posterior compartment (plantaris, gastrocnemius, and soleus) and intermuscular facial planes, predominantly in the anterior compartment (extensor digitorum longus) and lateral (peroneus longus). The patient was therefore suggested to undergo magnetic resonance imaging (MRI) for further evaluation.
FIGURE 1: Axial section STIR sequence of right lower limb.
Axial section STIR sequence of right lower limbs showing extensive intramuscular hyperintensities in the anterior compartment (extensor digitorum longus), lateral (peroneus longus), and superficial posterior compartment (plantaris, gastrocnemius, and soleus) and intermuscular facial planes, predominantly in the proximal part. There are no e/o any necrotic changes.

FIGURE 2: Axial section T2-weighted sequence of the right lower limb.
Axial section T2-weighted sequence of right lower limbs showing extensive intramuscular hyperintensities in the anterior compartment (extensor digitorum longus), lateral (peroneus longus), and superficial posterior compartment (plantaris, gastrocnemius, and soleus) and intermuscular facial planes, predominantly in the proximal part. There are no e/o any necrotic changes.
Coronal section STIR sequence of right lower limb showing extensive intramuscular hyperintensities in the anterior compartment (extensor digitorum longus), lateral (peroneus longus), and superficial posterior compartment (plantaris, gastrocnemius, soleus) and intermuscular fascial planes, predominantly in the proximal part. There are no ecoclastic changes.
FIGURE 4: Coronal section T2-weighted sequence of the right lower limb.

Coronal section T2-Weighted sequence of right lower limb showing extensive intramuscular hyperintensities in the science compartment (extensor digitorum longus), lateral (peroneus longus), and superficial posterior compartment (plantaris, gastrocnemius, and soleus) and intermuscular fascial planes, predominantly in the proximal part. There are no eco necrotic changes.

Treatment and follow-up

A course of the nonsteroidal anti-inflammatory drug (NSAID), namely, Indomethacin 75mg OD, and immunomodulators such as glucocorticoids, namely, Prednisolone 50mg OD were advised to the patient for three weeks with tapering of Prednisolone dosage after two weeks. A month after receiving the therapy, the patient underwent a follow-up examination. The patient’s symptoms had entirely subsided without a trace of the presenting complaints. The subsequent biochemical tests for inflammatory markers such as ESR, CK, and LDH readings had all returned to baseline.

Discussion

The SARS-CoV-2 coronavirus was formally declared a pandemic on March 11, 2020 [2,3]. There have been over 21 million cases of COVID-19 as of August 2020, reported worldwide, with over 800,000 COVID-19-associated deaths [4]. The possibility of developing a severe form of the illness is more so in older age groups and patients with comorbidities such as cardiovascular disease, diabetes mellitus, and obesity. Corticosteroids, mechanical ventilation, thromboembolic prophylaxis, and oxygen therapy are potential therapeutic options for COVID-19 patients who are experiencing acute symptoms [5]. Treatment guidelines, prevention measures, and presentations of COVID-19 are currently being updated. The SARS-CoV-2 is a single-stranded positive sense RNA virus that is coupled with a nucleoprotein inside a capsid made of matrix protein in spherical or pleomorphic excrional particles. In addition to the gastrointestinal system, urinary system (kidney and bladder), the pancreas, spleen, heart, and blood vessels, the Angiotensin-converting enzyme 2 (ACE2) receptor is highly expressed in lung epithelial cells [3,5]. Additionally, the central and peripheral neural systems, as well as diaphragm muscle, contains ACE2 receptors. [6] Angiotensin-converting enzyme 2 receptors are found in human cells and are recognized by this RNA virus. Viral release by cell apoptosis occurs after replication of the virus within human host cells [5,5]. Additionally, coronavirus induces an inflammatory response (including immunological responses, which are natural innate, and adaptive), which may lead to an overproduction of cytokines and eventually multi-organ destruction [5,5]. A patient infected with SARS-CoV-2 may present without any debilitating symptoms or with mild to moderate upper respiratory tract infection (URTI) or with acute respiratory distress syndrome (ARDS). Initially, it was primarily known to affect the respiratory system. It is now recognized that SARS-CoV-2 infection can cause a wide range of extrapulmonary signs. Gastrintestinal symptoms, dysfuction of the heart, kidney, and liver, acute coronary syndrome, dermatologic abnormalities, and neurologic sequelae are the extrapulmonary manifestations noted. Although myalgia is a prevalent clinical symptom in SARS-CoV-2 virus–infected individuals, early in the pandemic, additional musculoskeletal signs of COVID-19 were seldom reported [3-5]. Nevertheless, there have been more reports of rheumatologic problems and neuromuscular symptoms linked to the COVID-19 virus, the associated therapy, and course in the hospital as the number of patients and survivors increased globally [6].

COVID-19–associated musculoskeletal symptoms need to be carefully evaluated and managed effectively in order to avoid further complications such as rhabdomyolysis, which eventually leads to damage to vital organs such as kidneys. Imaging techniques such as ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI) can help in the diagnosis and evaluation of COVID-19–associated musculoskeletal symptoms and iatrogenic complications. In this article, we discuss the imaging characteristics and the mechanisms with which SARS-CoV-2 affects the musculoskeletal system. In particular, we review the various pathologies affecting the muscle. In extensive cohort studies, myalgia, which is characterized as muscle aches and pains, has been repeatedly observed in SARS-CoV-2 infection with a prevalence ranging from 11% to 50% [5,5]. Myophath and rhabdomyolysis have been mentioned in several case reports as occurring in SARS-CoV-2 infection. It can occur as both a late consequence of the infection or a presenting symptom. There have been...
a few isolated reports of COVID-19 affected patients with necrotizing autoimmune myositis. The mechanisms behind the involvement of the muscles in COVID-19 remain unclear. It has been shown that SARS-CoV-2 can propagate hematogenously and directly invade skeletal muscle by binding to the ACE2 receptor [6,7]. A distinct and more widely recognized notion of SARS-CoV-2 muscle involvement is immunological-mediated mechanisms, which are assumed to be an inflammatory response with immune cell activation and massive release of cytokines. Cytokines released from the muscle due to toxicity, injury resulting from the similarity between human muscle cells and viral antigens, and deposition of immune complexes are some of the hypothesized mechanisms of immune-mediated damage to the muscle [7].

Myositis is a general term for muscle inflammation and has been linked to viral illnesses such as Hepatitis, HIV, and influenza A/B, in addition to coronavirus. A side effect of myositis is myonecrosis and myoglobinuria which is due to muscle infection and obliterates myoglobin levels in the blood due to diaphragm failure, respectively. Diaphragm failure is a potentially fatal illness that can result in intracardiac congestion, compartment syndrome, and acute renal failure [8]. Myalgia and/or weakness and increased CRP levels have both been noted in individuals affected by COVID-19, which are typical clinical signs of myositis/thaldomycin [9]. Other illnesses causing muscle weakness or muscular skeletal symptoms such as neurological disorders like muscular dystrophies, multiple sclerosis (MS), amyotrophic lateral sclerosis (ALS), autoimmune diseases such as Graves’ disease, rheumatoid arthritis, thyroid conditions, and electrolyte imbalances have to be ruled out.

To establish a myopathic process and rule out analogs like motor neuron illness, electromyographic investigations like electromyography (EMG) and nerve conduction studies in muscle biopsies [10].

The preferred imaging technique is MRI, ideally using a 1.5 T or 3.0 T magnet and including fluid-sensitive multiplanar, anatomic sequences. Muscle edema is detected as a rise in signal intensity on short tau inversion recovery sequences (STIR) or T2-weighted sequence, which is suggestive of findings of myositis [11]. Hyperintense hypointense signal enhancement, heterogeneous hyperintense signal, and rim enhancement are examples of illness patterns [12]. Region of necrosis or the loss of typical muscle architecture may be visible in severe illness. The “stipple sign,” which consists of a region of non-enhancing muscle tissue with a surrounding rim of enhancement that contains enhancing fact within the muscle is a distinctive feature of myositis [13]. On gradient echo sequences, intramuscular hemorrhage can be seen as a T1 hyperintense signal or a blooming artifact [14].

A commonly accepted condition affecting the multiskeletal system in SARS-CoV-2 affected individuals needing ICU care is critical illness myopathy. It has been seen in association with coronavirus use in patients on ongoing treatment for COVID-19. Cortisol-based therapy is still the key treatment and recommendation for specific critically ill patients because of its strong anti-inflammatory and anti-fibrotic effects [15]. Thus, it is use of the differential diagnosis for muscle-skeletal trauma on MRI evaluation of SARS-CoV-2 affected individuals who are hospitalized. Symmetrical and widespread swollen or sudden fluid quadriceps are non-clinical manifestations of critical illness myopathy, which is a primary myopathy. This critical illness myopathy has non-specific imaging features of widespread muscular atrophy and edema. In contrast to the thalamomysitis/myonecrosis associated with COVID-19, there are no signs of necrosis in critical illness myopathy. In one research of critically sick COVID-19 patients done by Cabanes Martinez et al., the degree of spontaneous activity on electromyographic examinations was shown to be notably severe [16]. Patients with COVID-19 may develop dysfunction of the most principal muscle of the respirations the diaphragm. Dysfunction may occur as a result of phrenic nerve injury, potentially as a result of the implantation of chest support devices or critical illness myopathy and or maybe ventilator-induced diaphragm dysfunction. SARS-CoV-2 virus could theoretically cause direct neuromuscular involvement, which could result in diaphragm dysfunction. A recent autopsy investigation done by Shi Z et al. discovered the expression of angiotensin-converting enzyme 2 receptor in the SARS-CoV-2 virus and in the human diaphragm in a subgroup of COVID-19 patients who had diaphragm dysfunction [17]. Diaphragm dysfunction might cause respiratory problems and or makes it difficult to stop using ventilatory support in patients [15,18]. A rapid evaluation of diaphragm excursion is of utmost importance and is provided by the fluoroscopy sniff test, which is rapid and provided in real-time. Additional information is provided by ultrasound which demonstrate diaphragm muscle atrophy, examination of excursion with M mode, and the muscle thickening ratio with respiration. In the neck region, high-resolution ultrasound can also assess the phrenic nerve, which may help distinguish between neuropathic and myopathic causes of diaphragm failure. In COVID-19 patients with pronounced illness, long-term muscular sequelae like sarcopenia and cachexia are noted and have been well documented [19]. Muscle loss, also known as sarcopenia or myopathy, is mostly brought by age; however, inactivity and poor diet can also be contributing factors. Muscle wastage is caused by a persistent condition called cachexia. Foravitro and in-vivo muscle size can be MRI imaging signs of muscular atrophy, which are seen in sarcopenia and cachexia [21]. For the treatment of myositis, a variety of immunosuppressive and immunomodulatory therapeutic drugs are currently available. Glucocorticoids and immunosuppressants continue to be the first-line treatments; however, start and enough dosage can result in disease stabilization, strength recovery, and a reduction in inflammation. However, it’s important not to underestimate the negative effects of immunosuppressive therapy. Early addition or escalation of therapy should be prompted by refractory cases and extraocular symptoms, such as interstitial lung disease, heart involvement, etc. Treatment of difficult cases is dealt with by innovative treatment strategies that target particular immune pathways that show great promise to accurately anticipate the response to a particular treatment, it is also imperative to conduct an additional study into the pathophysiology of myositis. Together, these initiatives may, perhaps, lead to future advancements in myositis therapy [17].

Conclusions

COVID-19 and its predictable manifestations are generally recognized and studied extensively. The unusual symptoms and signs of the SARS-CoV-2 infection need a more careful and thorough examination. In our case report, we discuss the consequences of COVID-19 in the multiskeletal system, in particular, the muscles. MRI imaging can be very helpful in determining the involvement and characteristics of the multiskeletal disease associated with COVID-19. It can be used for both the initial diagnosis and the follow-up evaluation to estimate the progression of the illness and assess its recovery. Multiskeletal imaging may occasionally even serve as an indication of a previously undetected SARS-CoV-2 infection. Radiologists should be acquainted with the imaging findings, incidence, and etiology of COVID-19-related multiskeletal symptoms to interpret imaging reports and provide patients with the best intervention possible in an expeditious manner.

Additional Information

Disclosures

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