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Nonbacterial Myositis

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Abstract Infectious myositis is defined as an infection of a skeletal muscle. Infectious myositis is most commonly caused by bacteria; however, a variety of viral, parasitic, and fungal agents may also cause myositis. The pathogenesis of nonbacterial infectious myositis is via direct or hematogenous infection of the musculature or immune mechanisms. Symptoms typically include muscular pain, tenderness, swelling, and/or weakness. The diagnosis of the specific microbe is often suggested by the presence of concordant clinical signs and symptoms, a detailed medical and travel history, and laboratory data. For example, immunocompromised hosts have a heightened risk of fungal myositis, whereas the presence of a travel history to an endemic location and/or eosinophilia may suggest a parasitic cause. Definitive diagnosis requires detecting the organism by specific laboratory testing including serologies, histopathology, and/or cultures. Treatment entails antimicrobial agents against the pathogen, with consideration for surgical drainage for focal purulent collections within the musculature.

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Introduction

Infectious myositis is an infection of the skeletal musculature. Most commonly, infectious myositis is caused by bacteria, predominantly staphylococcal and streptococcal species [1•, 2]. Although pyomyositis was initially considered a "tropical disease," cases are well described in temperate regions. In these areas, cases appear to be rising in number, likely secondary to the aging of the population, increasing number of immunocompromised persons, and changing travel/migration patterns [3••]. In this review, nonbacterial infectious causes of myositis are discussed, including viruses, fungi, and parasites.

Pathogenesis and Clinical Presentation

The pathogenesis of infectious myositis involves invasion of the musculature via contiguous sites of infection or by hematogenous spread. Overall, infectious myositis is uncommon, largely because of the relative resistance of the musculature to infection [4]. When myositis (especially bacterial or fungal) occurs, it often does so in the setting of muscular damage from trauma, surgery, or vascular insufficiency, and/or an immunosuppressed state.

Viruses (eg, influenza), the most common cause of nonbacterial infectious myositis, often present as diffuse muscle involvement with myalgias, multifocal myositis,

and/or rhabdomyolysis. Viruses may affect the musculature directly by invasion or indirectly via immune mechanisms. Fungal myositis is less common, but has been described especially among immunocompromised patients and presents as either a single focal infection of the muscle or as part of disseminated disease. Finally, certain parasites (eg, toxoplasmosis, trichinosis) have a propensity for muscular involvement, often affecting several distinct muscle groups; the appropriate exposure history and/or travel history are important in diagnosing these cases.

The clinical presentation of nonbacterial infectious myositis depends on the number and the location(s) of muscles involved as well as host characteristics. In addition, the presence of signs and symptoms consistent with the pathogen are often helpful in pointing to the cause. The clinical presentation may be acute, subacute, or chronic in nature. Symptoms of myositis include localized or diffuse muscle pain, tenderness on palpation or with movement, swelling, and/or weakness. Myositis may mimic other conditions including muscle strains, hematomas, thromboses, osteomyelitis, or septic arthritis [5••]. The large muscles of the lower extremities are most commonly affected (eg, the quadriceps, calf, and gluteus muscle groups), perhaps as a result of strenuous exercise or trauma leading to muscle injury [5••, 6]; however, any skeletal muscle group may be involved.

Diagnosis

Establishing the cause of myositis is crucial in the management of cases. For myositis involving a focal area of the musculature (eg, an infectious collection on imaging), diagnostic testing should include sampling of the collection. A Gram stain as well as other microbiologic stains (eg, Gomori-Grocott methenamine silver stain for fungi), histopathologic examination, and cultures should be performed as suggested by the history. In cases of diffuse muscle involvement, the workup is largely based on the presence of other clinical symptoms and examination findings. For example, in cases in which a viral cause is suspected, standard viral testing should be ordered. In cases suggestive of a parasitic etiology, serologic and other laboratory data should be obtained as discussed below. A wide variety of nonbacterial infectious agents may cause myositis as shown in Table 1.

Causes of Nonbacterial Myositis

Viral Agents

Viruses are the most common etiologies of nonbacterial infectious myositis cases in the United States and the rest of

Table 1 Nonbacterial causes of infectious myositis

- Viruses
  - Adenovirus
  - Cytomegalovirus
  - Dengue virus
  - Enteroviruses (coxsackie A/B viruses and ECHO viruses)
  - Epstein-Barr virus
  - Hepatitis A, B, and C viruses
  - Herpes simplex virus
  - Human immunodeficiency virus
  - Human T-cell lymphotropic virus-1
  - Influenza A and B viruses
  - Mumps
  - Parainfluenza virus
  - Parvovirus B19
  - SARS-coronavirus
  - Varicella-zoster virus
  - West Nile virus
- Fungi
  - *Aspergillus* spp
  - *Blastomyces dermatitidis*
  - *Candida spp*
  - *Coccidioides immitis and posadasii*
  - *Cryptococcus neoformans*
  - *Fusarium* spp
  - *Histoplasma capsulatum*
  - Mucormycosis
  - *Pneumocystis* (*carinii*) *jiroveci*
  - *Sporothrix schenckii*
- Parasites
  - Helminths:
    - Nematodes (tissue roundworms)
      - *Trichinella* spp
      - *Haycocknema perplexum*
      - *Toxocara canis*
      - *Onchocerca volvulus*
    - Cestodes (intestinal/larval tapeworms)
      - *Taenia solium*
      - *Echinococcus* spp
    - Trematodes (flukes)
      - *Schistosoma* spp
  - Protozoa:
    - *Toxoplasma gondii*
    - *Entamoeba histolytica*
    - *Sarcocystis* spp
    - *Spirometra mansonoides*
    - *Trypanosoma cruzi*
    - *Plasmodium* spp
  - *Microsporidia* spp (*Annicaliia, Trachipleistophora, Pleistophora* spp)

ECHO enteric cytopathogenic human orphan (viruses); *SARS* severe acute respiratory syndrome

the developed world. Clinical syndromes include generalized myalgias, polymyositis, and/or rhabdomyolysis. Influenza viral infections are the most common agents described; however, a wide variety of other viruses have been implicated (Table 1).

Influenza

Influenza viral infections typically present with fever, cough, and rhinorrhea. Myalgias may also occur as part of the initial symptom complex and are typically diffuse and self-limited in nature. Later in the course of illness, patients may develop myositis, first described in 1957 [7] and referred to as "acute benign myositis." In a large case series of children diagnosed with influenza, rates of benign acute childhood myositis among influenza A and influenza B cases were 5.5% and 33.9%, respectively [8•].

Signs and symptoms of myositis include pain, tenderness, and swelling of the musculature typically located in the gastrocnemius and soleus muscles; other muscles may also be involved. Refusal to walk is a common finding, particularly among children [8•, 9, 10••]; however, muscle strength is usually normal on physical examination. Symptoms of myositis usually begin a mean of 3 days (range 0–18) after initial influenza presentation. Myositis can be differentiated from myalgias by its later occurrence, more localized location, and increased severity [8•].

Influenza-associated myositis typically occurs among children (although adults, including the elderly, may also develop myositis) [8•, 10••, 11]. The reason that children are at higher risk for this condition may be because immature muscle cells are more permissive to infection [10••]. Myositis also appears to preferentially involve boys (2:1 ratio) and is more often associated with influenza type B versus A, perhaps because of the presence of a glycoprotein unique to B strains that renders the virus more myotropic [10••]. A case of acute benign myositis caused by the novel H1N1 (2009) strain was recently reported [12•].

The exact cause of influenza-associated myositis is unclear (direct viral invasion vs an immunologic mechanism); however, influenza has been isolated from muscle tissues, suggesting that direct viral invasion into the muscle fibers does occur in at least some cases [13, 14]. Other findings on biopsy include edema and focal infiltration of polymorphonuclear and mononuclear cells.

Diagnosis of influenza-associated myositis is usually made by the clinical presentation, including the presence of influenza activity within the community, and by detecting the influenza virus using rapid or polymerase chain reaction (PCR) testing of nasopharyngeal specimens. Creatine phosphokinase levels are usually elevated, as are other muscle-associated enzymes. Recognition of influenza as the etiology is important because this virus has also been linked to the development of rhabdomyolysis; as such, close monitoring for its occurrence and the initiation of early therapy is recommended to reduce complications [9]. Furthermore, diagnosis of influenza as the cause may prevent unnecessary diagnostic and therapeutic interventions.

Symptoms typically resolve within 1 week, but occasionally last up to 1 month [10••]. Treatment involves symptomatic care. Most cases of myositis present after the 48- to 72-hour recommended window for initiation of antiviral medications; hence the utility of antiviral agents (eg, neuroaminidase inhibitors) for the treatment of influenza myositis is unknown.

In addition to benign cases of muscle involvement, influenza viruses can cause life-threatening rhabdomyolysis, which may be complicated by renal failure [10••]. Of note, in a review of rhabdomyolysis cases, influenza was the most common isolated cause [15]. Unlike acute benign myositis, rhabdomyolysis is more commonly associated with influenza type A versus B, and is more commonly described among girls. Rhabdomyolysis may occur in association with both the seasonal and novel 2009 H1N1 strains [12•, 15].

Coxsackievirus

Pleurodynia syndrome is a well-recognized clinical condition due to group B (rarely group A 4, 6, 9, and 10) coxsackievirus infections and, less frequently, enteric cytopathogenic human orphan (ECHO; 1, 6, 9, 16, and 19) viruses [16–18]. Manifestations typically consist of paroxysmal, sharp thoracic and upper abdominal muscle pains (especially in the intercostal regions), with localized tenderness and fever [18]. Headache and sore throat may also be present. Cases are typically noted in the summer and fall months.

The clinical course is usually self-limited with symptoms lasting about 5 days; however, recurrences have been described in up to a quarter of cases. Although direct viral invasion is the proposed pathogenic mechanism, evidence is currently lacking regarding the pathogenesis of this condition. Diagnosis typically relies on the classic clinical presentation. Therapy is symptomatic; the disease typically resolves in several days.

Coxsackieviruses (A9, B2, B6) and echoviruses (9, 11) may also cause myositis in other skeletal muscles outside the torso, as well as rhabdomyolysis [15, 19, 20]. Clinical findings may include fever, tenderness, edema, weakness, and hypotonia. Muscle enzymes are often elevated, and myoglobinuria is present. The pathogenesis of myositis is unclear, but muscle biopsies have shown viral-like structures. Most cases recover uneventfully. Finally, coxsack-

ievirus may cause myocarditis, especially among newborns [21].

Diagnosis of coxsackievirus is usually made on the basis of the clinical picture along with laboratory testing: serologic evaluation for acute coxsackievirus infection or culture for the virus from pharyngeal or fecal specimens. Muscle biopsies are not typically advocated, but if performed, viral antigens can be detected by immunofluorescence or the viral RNA by PCR. As with most viral-associated myositis cases, therapy is symptomatic.

Human Immunodeficiency Virus

Persons infected with HIV may develop a myriad of muscle disorders, including polymyositis, with findings of myalgias, weakness (involving predominantly the proximal muscles), and elevated muscle enzyme levels [22]; the presentation mirrors that of idiopathic polymyositis. HIV patients may have an increased susceptibility to myositis because of the combination of underlying immunodeficiency and the presence of concurrent conditions or medications (eg, zidovudine) that may contribute to muscle injury. Polymyositis may represent the initial presentation of HIV, which leads to its diagnosis. Viral antigens have been found in the $CD4^+$ T lymphocytes surrounding the muscle fibers, but not specifically within the muscle fibers themselves, suggesting that the pathogenesis may be immune mediated [22, 23].

In addition to myositis as the initial presentation of HIV, patients may develop other forms of muscle disease throughout their course, including myopathies associated with HIV itself, antiretroviral medications (eg, nucleoside-related mitochondrial myopathy), wasting, inclusion-body myositis, nemaline myopathy, diffuse infiltrative lymphocytosis syndrome, vasculitic processes, myasthenic syndromes, and opportunistic infections and malignancies involving the skeletal musculature [24]. Finally, cases of rhabdomyolysis associated with HIV infection have been reported [25].

Within the diagnostic workup of a possible viral-associated myositis, HIV testing should be considered. Among known HIV patients, the workup of muscular disease includes a careful history and physical examination, and consideration for electromyographic studies and muscle biopsy. Management of cases involves supportive care and treatment of the specific etiology of the muscle disease identified; cases that are determined to be autoimmune-related may benefit from corticosteroid therapy.

Other Viruses

Human T-cell lymphotrophic virus type (HTLV)-1 is the cause of adult T-cell leukemia/lymphoma and tropical spastic paraparesis/HTLV-1-associated myelopathy. HTLV-1 is a known cause of polymyositis in locations of endemicity including the Caribbean, Africa, Japan, Melanesia, and the southern United States. Like HIV, the pathologic mechanism is thought to be immunologic [23, 26].

Hepatitis B and C viruses were linked in case reports to the development of polymyositis [27, 28]. Adenovirus may cause myocarditis, myositis, and rhabdomyolysis [29, 30]. Parvovirus B19 leading to myositis was described in conjunction with fever and a diffuse rash, including involvement of the cheeks [31]. West Nile virus may cause myositis in addition to neurologic findings, although further information on this occurrence is needed [32]. Finally, tropical viral diseases may also be associated with myalgias and/or myositis, such as with dengue [33, 34]. Other viral agents linked to myositis are listed in Table 1.

Rhabdomyolysis may be caused by a variety of viral agents, including influenza (the most common etiology), parainfluenza, enteroviruses (Coxsackie, ECHO), adenovirus, severe acute respiratory syndrome (SARS)-coronavirus, HIV, herpes viruses (varicella, herpes simplex, Epstein-Barr, cytomegalovirus), parvovirus B19, dengue, and West Nile virus [15, 35]. Symptoms are typically myalgias, weakness, muscle tenderness, and edema, and laboratory findings show high levels of creatine kinase with the presence of myoglobinuria which may be complicated by acute renal failure.

Diagnosis of viral-associated myositis and/or rhabdomyolysis is suggested by the presence of concurrent, appropriate clinical symptoms and the performance of serologic studies or cultures of nasopharyngeal, respiratory, or stool specimens. Rarely, muscle biopsies have been performed in an attempt to identify a viral etiology by immunofluorescent or PCR studies, electron microscopy, or viral isolation.

Fungal Agents

Fungal involvement of the musculature is uncommon, but it was described in case reports and often presents similarly to bacterial myositis. Most cases have occurred among severely immunocompromised patients, but also have been reported among immunocompetent persons. Both the setting of an immunosuppressed state and evidence of other sites of fungal involvement may suggest a fungal etiology. Biopsy with culture is usually necessary to establish the diagnosis.

Candidiasis

The most commonly reported cause of fungal myositis is *Candida*. Most cases occur among patients with significant

immunosuppression, especially in the setting of chemotherapy and broad-spectrum antibiotic use [36, 37]. A triad of fever, rash, and muscle tenderness among candidemic patients suggests myositis [38]. Diffuse, multiple microabscesses, or larger, focal fungal abscesses of the muscles may occur; the lower extremities are most commonly involved. The most common *Candida* species to involve the musculature has been *C. tropicalis*, but a variety of species have been implicated (eg, *Candida krusei, Candida albicans*) [36, 37, 39].

Diagnosis is typically confirmed by a muscle biopsy, with histopathology showing budding yeast and pseudohyphae and growth of the organism on fungal culture. Imaging with MRI, CT, or ultrasound can be used for localization of the biopsy site [39]. Fungal blood cultures may be positive. Therapy is with antifungal agents such as amphotericin B, an azole, or an echinocandin. In addition, focal infectious collections within the musculature, especially if large, should be surgically drained.

Cryptococcosis

Cryptococcus neoformans may disseminate and cause myositis after inhalation of the organism. Cases may occur in the setting of disseminated cryptococcosis or as focal muscular infection [6, 40]. Most cases occur in the setting of HIV/AIDS, although cases in transplant recipients and patients with leukemia/lymphoma have been reported [6, 40, 41]. Pain and swelling in the muscles of the lower extremity is the most common presentation. Muscle biopsy demonstrates the presence of the fungus. Diagnosis may be supported by a positive cryptococcal serum antigen; an estimated 60% have positive blood cultures [40]. Cryptococcal myositis should prompt investigation for infection at other sites, including the central nervous system (CNS), via performance of a lumber puncture. Treatment is with amphotericin B and flucytosine for multifocal disseminated disease or with concurrent cryptococcemia. Fluconazole has been used for localized muscular disease alone. Surgical debridement of focal purulent collections should be performed [40].

Histoplasmosis

Histoplasma capsulatum is a fungal infection acquired via inhalation of the dimorphic fungal microconidia; dissemination may occur, including muscular involvement. Cases of myositis have occurred in AIDS patients and other immunocompromised persons, including those with autoimmune disease receiving immunosuppressive agents [42–44]. Diagnosis has been made by biopsy, with histopathology showing ovoid yeast cells and cultures yielding growth of the fungus. *Histoplasma* antigen testing may be positive [43]. Cases in the literature most commonly were treated with amphotericin B; large focal collections can also be surgically drained.

Aspergillosis

Aspergillus spp, like other causes of fungal myositis, occurs predominantly among immunocompromised persons. Similar to *Candida* and *Cryptococcus*, myositis has been described as part of disseminated disease or as a focal infection [45–47]. Diagnosis is by biopsy with fungal culture. The accuracy of fungal antigen testing (eg, galactomannan test) is unknown in myositis cases [46]. Treatment of *Aspergillus* is typically with voriconazole or amphotericin B and debridement of any necrotic tissue resulting from vascular invasion and thrombosis.

Other Fungi

Other less common etiologies of fungal myositis include *Coccidioides* spp [48], *Pneumocystis* (*carinii*) *jiroveci* [49], mucormycosis [50], *Fusarium* spp [51], *Sporothrix schenckii* [52], and *Blastomyces dermatitidis* [53] (Table 1).

Parasitic Agents

A variety of parasitic infections may encyst in the musculature. For parasitic infections that are acquired abroad, a residence/travel history is an important component in diagnosing these cases. In addition, an exposure history of relevant food ingestion and the presence of eosinophilia may suggest a parasitic etiology of myositis. The most commonly reported causes of parasitic myositis in the United States include the helminthic infections of *Trichinella* spp (trichinosis) and *Taenia solium* (cysticercosis). Protozoa such as *Toxoplasma gondii* (toxoplasmosis), as well as *Microsporidia* spp, are also causative agents of nonbacterial myositis. Other parasites that are notable causes of myositis in endemic areas are shown in Table 1.

Trichinosis and Other Nematode Infections

Trichinella spp, most commonly *Trichinella spiralis*, can incidentally infect humans after ingestion of undercooked meats, such as pork and wild game meat (eg, bear, wild boar, cougar), and larvae may subsequently encyst within striated muscles. Although most infections are subclinical, gastrointestinal symptoms followed by systemic manifestations may occur, especially after large inoculums of larvae are ingested. Fevers and symptoms consistent with myositis (myalgias, swelling, and muscle weakness) initially occur in the extraocular muscles, followed by other muscles of the head and neck [54]. Any striated muscle can subse-

quently become involved. The presence of a rash, periorbital edema, diplopia, and/or conjunctival hemorrhage supports the diagnosis. Rarely, cases have included severe proximal muscle weakness simulating polymyositis [55]. Signs of myositis peak 2 to 4 weeks after infection and then begin to wane, although some patients may have muscle aches for several weeks. Infection is typically self-limiting; however, some patients develop myocarditis and other complications from the encysted larvae.

Diagnosis is by the clinical history (including dietary intake of potentially undercooked meat) and the presence of eosinophilia and elevated muscle-related enzymes. Serologic testing with the enzyme-linked immunosorbent assay (ELISA) can yield the diagnosis; antibodies are usually detectable by 2 to 4 weeks after infection, and a rising titer is suggestive of an acute infection. Radiographs may show calcified cysts later in the course of the infection. Diagnosis can also be confirmed by muscle biopsy (ideally obtained from a painful muscle near its insertion site) showing coiled larvae, cysts, and eosinophils, but this procedure is usually not necessary. PCR testing of the biopsy can also be used for confirming the diagnosis and determining the species of *Trichinella* involved. Treatment is with mebendazole or albendazole, which should be administered early in the course of the infection, ideally in the gastrointestinal phase before muscle encystment occurs [56]. Life-threatening forms of trichinosis, such as severe myositis, myocarditis, and neurologic complications, are often treated with a combination of albendazole and corticosteroids [54].

Another nematode that may cause myositis is the novel parasite, *Haycocknema perplexum* of the Muspiceoidea superfamily [57, 58]. Although cases have not been described in the United States, reports from Australia and Tasmania have been published. Typically, cases present with proximal muscle weakness consistent with polymyositis. Laboratory findings include eosinophilia and elevated creatine kinase levels. Diagnosis is made by muscle biopsy showing the nematode, and therapy is with albendazole. Of note, the use of steroids in these cases may result in clinical deterioration.

Cysticercosis

Ingestion of eggs of the pork tapeworm, *Taenia solium*, in contaminated food or water can lead to cysticercosis. Cases in the United States are typically acquired abroad and are most commonly found in states bordering Mexico. After ingestion, the eggs develop into invasive larvae that penetrate the intestinal wall and disseminate with a particular tropism for the CNS, subcutaneous tissues, and muscles. The disease invokes only a mild inflammatory response, hence, it is often silent unless the cysticerci begin to degenerate, causing seizures or the formation of subcutaneous nodules. Muscular involvement is uncommon and is usually asymptomatic; lesions imitating tumors or myositis with myalgias and weakness due to the inflammatory reaction around dying cysts have been reported [59, 60]. An uncommon form of muscle involvement called "pseudohypertrophic myopathy" is caused by multiple cystic lesions, particularly involving the calf muscles [61].

Imaging (eg, MRI, CT, and ultrasound) can suggest the diagnosis by showing a clear cyst with a scolex. Radiographs may demonstrate calcified cysts within the musculature that have a "puffed rice" or "spindle-shaped" appearance. Serologic testing with ELISA using blood or cerebrospinal fluid specimens can aide in the diagnosis. Biopsies may be performed, revealing cysts, but are usually not necessary.

Treatment of muscle involvement with viable cysts is with albendazole or praziquantel; cases of myositis without evidence of viable cysts and which are asymptomatic may not require therapy. Large intramuscular lesions have been excised surgically and treated with antihelminthic medications [59]. Patients with muscular cysticercosis should undergo CNS imaging, because most cases have concurrent brain involvement.

Toxoplasmosis

This infection is acquired via ingestion of undercooked meat (usually pork or lamb) containing *Toxoplasma gondii* tissue cysts, or by ingestion of food items contaminated with oocysts from cat feces. The disease may be acquired worldwide, including in the United States. Acute infection among immunocompetent persons is usually asymptomatic, but may present as a self-limited, heterophile-negative, mononucleosis-like illness consisting of fever, cervical adenopathy, myalgias, and malaise. Occasionally, acute disease may result in polymyositis and/or myocarditis among both immunocompetent persons and those with T-cell deficiencies, including those receiving corticosteroids [62]. Immunocompromised patients, especially those with AIDS or with low CD4 T^{+} cell levels, may develop reactivation disease involving the musculature [63, 64].

Symptoms of polymyositis mirror those of autoimmune disease and include weakness, wasting, fasciculations, swelling, and myalgias. Additional symptoms due to systemic toxoplasmosis may include fever, encephalitis, and multiorgan failure. Diagnosis is made based on a compatible clinical history along with serologic testing. Acute infection is demonstrated by a positive IgM; those with reactivation have high IgG titers. Serum creatine kinase levels are usually elevated. Muscle biopsy may show muscle fiber destruction and interstitial infiltrate. The intracellular parasite of *Toxoplasma* may be seen by immunocytochemistry or microscopy; isolation of the

organism has been achieved by tissue culture. Therapy for toxoplasmosis with sulfadiazine and pyrimethamine should be administered early in cases with myositis. Steroids have also been used in the treatment, especially among cases diagnosed later in the disease course [63, 65].

Microsporidiosis

Microsporidia species are found widespread in nature; the route of transmission to humans is unclear, but may occur via insect bites or ingestion of contaminated water sources or undercooked meats [66, 67]. Microsporidiosis is an increasingly diagnosed condition, mirroring the rising number of immunosuppressed persons [67]. AIDS patients with CD4 cell counts of less than 100 cells/mm^3 are at particular risk for these infections. Receipt of corticosteroids and tumor necrosis factor antagonists (eg, infliximab) also appears to increase the risk of microsporidiosis [66].

Myositis may occur as part of disseminated infections with *Trachipleistophora hominis, Trachipleistophora anthropophthera, Pleistophora ronneofiei, Annicaliia (formerly Brachiola and Nosema) algerae, Annicaliia vesicularum*, and *Annicaliia connori* [66, 67]. Muscle involvement may include myocarditis or involvement of the skeletal muscles. Clinical symptoms include muscle pain, weakness, wasting, and fever. Creatine kinase levels are often elevated. Diagnosis is by muscle biopsy with light and electron microscopic examination. Treatment is with albendazole. The use of combination therapy consisting of albendazole with itraconazole has been reported for *A. vesicularum* myositis. Patients with HIV should also receive antiretroviral therapy because immune reconstitution may aid in disease resolution.

Other Parasites

Several other parasitic agents may cause myositis, including *Trypanosoma cruzi* (Chagas' disease) [68], *Sarcocystis* spp [69], *Toxocara canis* (visceral and ocular larva migrans due to the dog tapeworm) [70], *Schistosoma* spp (schistosomiasis) [71], *Echinococcus* spp (hydatid disease) [72], *Entamoeba* spp (amoebiasis) [73], *Spirometra* spp (sparganosis) [74], *Plasmodium* spp (malaria) [75], and *Onchocerca volvulus* (river blindness) [76], as shown in Table 1.

Noninfectious Myositis

In addition to infectious causes, a wide range of noninfectious etiologies should be considered in the differential diagnosis among patients presenting with myositis-like symptoms. These include the idiopathic inflammatory myopathies of polymyositis, dermatomyositis, and inclusion body myositis. Myopathies may also occur with autoimmune/collagen vascular diseases, malignancies, genetic disorders, endocrine diseases, and metabolic/electrolyte/nutritional disorders [5••, 77•]. In addition, several medications, including lipid-lowering drugs (eg, 3-hydroxy-3-methylglutaryl-coenzyme A [HMG-CoA] reductase inhibitors), are well-described causes of myositis [78].

Conclusions

Nonbacterial infectious myositis may be caused by a variety of viral, parasitic, and fungal pathogens. The pathogenesis of myositis includes both direct infection of the muscle and immune-mediated injury. Viral myositis is most often caused by influenza viruses and has a predilection for children. Fungal myositis is uncommon, and in temperate areas, most often occurs among immunosuppressed patients. Parasitic myositis should be considered in the setting of a compatible exposure history and/or concurrent eosinophilia. Diagnosis is based on the clinical picture and laboratory and radiologic information, and is confirmed by identifying the causative organism via serology, histopathology, or culture data.

Disclosure The content of this publication is the sole responsibility of the author and does not necessarily reflect the views or policies of the NIH or the Department of Health and Human Services, the DoD or the Departments of the Army, Navy or Air Force. Mention of trade names, commercial products, or organizations does not imply endorsement by the US Government. This work is original and has not been published elsewhere.
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