Morgellons Disease and Its Association with Spirochetes and Lyme Disease

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Keywords: Morgellons disease, Spirochetes, Lyme disease

INTRODUCTION

Morgellons disease is a rare disease with a previously mysterious etiology and pathogenesis. It is characterized by the presence of multi-colored filaments that lie under, are embedded in, or project from skin. The filaments can be white, black, or brightly colored [1]. It was previously considered to be a delusional disorder due to its similarity to delusions of parasitosis or delusional infestation described many years ago [2,3]. This constellation of symptoms has not been well studied in different populations, however, a study in North California found a prevalence of 3.65 per 100,000 for Morgellons disease, with Caucasian and female predominance. Its main symptom was stated to be development of fibres or materials from the skin, with or without cutaneous lesions, and it was found to significantly affect the health-related quality of life of the patients [4].

While the cause of Morgellons disease has not been fully established, there have been recent serological and clinical evidence linking it to Lyme borreliosis, hence it is thought to be a true somatic disease [5,6]. Morgellons disease parallels bovine digital dermatitis (BDD), an infectious disease of cattle, characterized by dermatitis and papillomatous lesions of the skin around the coronary band in the hooves of livestock. Livestock with BDD have been reported to be serologically reactive to the antigens of Borrelia burgdoferi [7]. This is the organism mainly responsible for Lyme disease, a spirochetal infection transmitted through the bite of infected ticks. This prompted the investigation into the possible link between spirochete and Morgellons. Furthermore, some symptoms present in patients with Morgellons disease often resemble those of Lyme disease, such as fatigue, arthralgia, and neuropathy [7,8].
Some symptoms that have been reported in Morgellons disease include crawling sensations under the skin; slow-healing lesions appearing suddenly; hyper pigmented scars when lesions heal; severe itching; seed-like objects or specks, in lesions or on intact skin; fine, thread-like fibres of varying colors in lesions and intact skin; lesions containing thick, translucent fibres; and a sensation of something trying to penetrate the skin from the inside out [8]. Filaments in Morgellons disease have been found to originate from epithelial cells, from the Stratum Basale, and from the root sheath of hair follicles, similar to BDD [7]. This points to the fact that the filaments may be of cellular origin, and not as a result of foreign materials such as textile fibre.

The fibers in Morgellons lesions are most likely spirochetes in their vegetative form, long and fibrous and characterized by a crest spiraling along the surface. Spirochetes have the ability to persist even in hostile environment and can adopt different sizes and shapes. They can form round bodies, L-form bacteria, micro colonies or biofilms-like aggregates, which remarkably change the response of *Borrelia* to hostile conditions and make it antibiotic resistant [9]. These fibres often require magnification of 50× or more to be seen, and at that magnification they can be mistaken for textile fibres [10].

**HISTORY OF MORGELLONS**

The two-year old son of Mary Leitao, a former laboratory technician suffered from dermatological lesions that contained multi-colored fibres upon magnification with a microscope. After dismissal from numerous doctors, she named the disease her son was suffering from Morgellons, due to its resemblance to a dermatological malady described in the 1600s [11]. The name came from a letter written in 1674 by Sir Thomas Browne, an English physician. The letter contained a brief description of a skin disease characterized by “outbreaks of harsh hairs” on the backs of French children [12]. However, the accounts by Browne and other physicians who described similar diseases were likely referring to a heterogeneous group of skin conditions different from the one referred to as Morgellons today. The diseases described previously occurred primarily in childhood and were often associated with cough and convulsions [12].

Mary Leitao founded the Morgellons Research Foundation in 2004 to raise awareness and funding for research into what she considered a disfiguring and disabling condition. The foundation believes that it is a new infectious disease that will be confirmed by future research. Since then, the disease has been publicly debated and has enjoyed wide media coverage. This has been blamed for the increase in number of self-diagnosed Morgellons disease sufferers [11]. A majority of health professionals, including most dermatologists, still regard Morgellons as a manifestation of other known medical conditions such as delusional parasitosis and believe any fibres found are exogenous, from items such as clothing [4,8,13].

**DISTRIBUTION**

Not many studies have been carried out on the epidemiology of Morgellons disease. In a cohort of 1000 North Americans with Lyme disease, 6% were diagnosed of Morgellons [14]. It is commoner in Caucasians, females, and those aged 45-60 years [4,8,14]. In 2017, the first case of Morgellons disease in Korea was reported in a 30-year-old woman. She presented with a 2-month history of pruritic erythematous patches and erosions on the arms, hands, and chin [15]. Most patients have also been found to have tickborne co-infections and to present with disseminated Morgellons disease [4,14]. Female predominance in Morgellons has been thought to be a result of the fact that females are meticulous when it comes to dermatological care, and are therefore more aware of any skin changes. Secondly, an exaggerated response to infections typically seen in women may explain the female predominance [14]. While no case of death directly linked to the disease has been reported, it has been found to cause significant physical and psychological distress to sufferers [8,10].

**CLINICAL ASPECTS OF MORGELLONS DISEASE**

Symptoms reported by patients with Morgellons disease include: emergence of materials from the skin, crawling sensations under the skin; slow-healing lesions appearing suddenly; hyper pigmented scars when lesions heal; severe itching; and a sensation of something trying to penetrate the skin from the inside out [4,8]. Also, musculoskeletal symptoms, fatigue, insomnia, cognitive impairment, depression, hypothyroidism, vision change, unexplained weight gain and anxiety have been reported among patients with MD [4,14].

A standardized guideline has not yet been established for the diagnosis of MD, however, Middelveen *et al* [16] proposed a diagnostic criteria as follows:

1. Primary features (Must include the following):
   - A Multicolored filaments embedded within or protruding from the skin
2. Secondary features (May include one or more of the following):
   A. Development of calluses
   B. Ulcerative lesions
   C. Papules
   D. Burning, itching, stinging, biting
   E. Hair loss
   F. Atypical hair/nail production
   G. Dry appearance with or without flaking skin
   H. Oedema
   I. Hyper- or hypo-pigmentation from scarring
   J. Hypertrophic scarring
   K. Excoriations
   L. Slowly healing lesions
   M. Aging skin

MD has also been classified into early localized, early disseminated, late localized and late disseminated stages based on the duration and location of the MD lesions. Early localized MD are those with lesions/fibers present for less than three months and localized to one area of the body such as the head, trunk or extremities. Early disseminated MD are those with lesions/fibers present for less than three months and involving more than one area of the body. Late localized lesions were those present for over six months and localized to one area of the body, while late disseminated MD are those with lesions/fibers present for more than six (6) months and involving more than one area of the body [6].

MD has also been classified as Mild (Stage A), moderate (Stage B) and severe (Stage C) MD based on unique histopathological patterns seen. Stage A lesions demonstrated minimal immune infiltrates and disorganization of cells; macrophages were not present, and haemorrhage was negligible. Extracellular isolated spirochetes and intracellular staining of keratinocytes in the lower epidermis was occasionally seen. Stage C lesions demonstrated positive staining of keratinocytes in the stratum basale and stratum spinosum and positive intracellular staining of macrophages for Borrelia. Aggregate Borrelia colonies were frequently seen, haemorrhage was frequent, and intracellularly stained fibroblasts were occasionally seen. Stage B lesions demonstrated a pattern intermediate between Stages A and C [16].

EVIDENCE OF LINKAGE WITH SPIROCHETES AND MORGELLONS DISEASE

Previous studies have shown the linkage between MD and sprochaetal infection, specifically Borrelia burgdorferi (Bb). Spirochetes was previously detected on light microscopy, however, the detection of borrelial species by culture, immunofluorescent staining, electron microscopy or PCR is currently valid proof in the presence of MD symptoms [17]. Savely and Striker (2010) in their study explored the link between Morgellons disease and Lyme disease, reported that 96.8% of their subjects with MD either tested positive for Lyme disease on Western blot or were clinically diagnosed, also, many had positive tests for coinfecting tick-borne illnesses. In addition, the distribution of the Lyme disease patients and Morgellons patients were quite similar [8]. Fesler et al I their study on 60 North Americans with MD found that all of them were positive for Bb infection. Similarly, tickborne coinfections such as Babesia spp (62%), Bartonella and Rickettsia (25% each), Ehrlichia (15%) and Anaplasma (10%) were found in patients with MD [14]. In another study, the presence of spirochetes was confirmed by multiple testing modalities, including culture, histology, anti-Bb immunostaining, electron microscopy, PCR and in situ Bb DNA hybridization, using dermatological tissue, blood and vaginal secretions [6].

CONCLUSION

Chronic itching, skin eruptions, crawling sensations, slow healing lesions, hyperpigmented scars. Anxiety and depression have been observed in some MD patients. MD has four stages: early, disseminated, and late. Lesions/fibers less than three months old. MD affects numerous organs early on. More than six (6) months old lesions/fibers affect many body regions. Mild, moderate, or severe MD (Stage C). Little or no immune infiltrates cell instability or macrophages. Spirochetes and keratinocytes Borrelia-positive keratinocytes and macrophages. Bloody fibroblasts and Borrelia colonies were prevalent. Borrelia burgdorferi infection linked to MD (Bb), there are currently Borrelia species detection methods. Savely and Striker claim 968 Morgellons patients had Lyme illness (2010). Similar to patient distribution, BC was identified in 60 North Americans with MD. Tickborne infections like Babesia (62%), Bartonella (25%), Rickettsia (15%), and Anaplasma (10%) were found in MD patients. In situ Bb DNA hybridization and PCR were used to find them.

REFERENCES


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