Mycoplasma infection and muscle pain: fact or fantasy?

Infectious agents, ranging from viruses to bacteria and fungi, have at one time or another been blamed for causing a variety of musculoskeletal, and associated, conditions, including chronic fatigue. One group of organisms, the Mycoplasmas, are currently receiving a great deal of attention in this regard.

According to Nicolson (1998) Mycoplasma infections have been observed in approximately 70% of fibromyalgia syndrome (FMS), 60% of chronic fatigue syndrome (CFS), as well as in 50% of Gulf War Illness and rheumatoid arthritis patients. Many of these patients were found to be infected by principally one species of Mycoplasma: *M. fermentans*. Root-Bernstein (1993) explains some of the characteristics of this odd bacteria:

> *Mycoplasma is a genus name for [approximately] 50 different species of bacteria. Mycoplasmas differ from most other bacteria in being relatively small and lacking an outer cell wall. They are often among the most difficult bacteria to isolate. They can cause a range of disease manifestations, including pneumonia, when present in the lungs, and proctitis, when they infect the rectum. Animals infected with Mycoplasmas often become immune suppressed.*

Mycoplasmas are primitive classes of bacteria that have the ability to incorporate into their own surface structures parts of host cell membranes that contain important host membrane antigens, creating the opportunity for autoimmune responses (Baseman & Tully 1997).

Nicolson et al. (2002) offer these further observations about the Mycoplasmas:

> *In CFS patients we have found that chronic infections are a rather common feature of the illness. Previously we studied American and European CFS patients and found that most had Mycoplasmal infections. .. When we examined the incidence of particular Mycoplasmal infections in CFS, we found that most patients had multiple infections (two or more species of Mycoplasma), which were for the most part combinations of M. fermentans and other Mycoplasma species… Mycoplasmas are found commonly in the oral cavity, urogenital tract and as symbiotic gut flora, but some species can cause acute and chronic illnesses when they penetrate into the blood vascular system and systemically colonize organs and tissues. For example, M. penetrans, M. fermentans, M. hominis and M. pirum can enter a variety of tissues and cells and cause systemic signs and symptoms. Mycoplasmas have also been shown to have a complex relationship with the immune system. They are very effective at evading host immune responses, and synergism with other infectious agents has been seen.*

The recommended treatment (Nicolson et al. 2000, 2002; Nicolson & Nicolson 1995) for confirmed Mycoplasmal blood infections in FMS or CFS patients is long-term antibiotic therapy, usually involving multiple 6-week cycles of doxycycline (200–300 mg/day) together with a number of other antibiotics, for up to a year. They justify this protocol as follows:

> *Multiple [antibiotic] cycles are required, because few patients recover after only a few cycles, possibly because of the intracellular locations of the infections, the slow-growing nature of these microorganisms and their inherent insensitivity to antibiotics. We now recommend that patients who have been diagnosed with [Mycoplasmal] blood infections receive continuous oral antibiotics for at least 6 months before using the 6-week cycles of treatment.*

Nicolson (1998) also recommends nutritional support for the immune system, as well as replenishment of gut bacteria, when treating Mycoplasma infection with antibiotics. But there is also a powerful contrary view. Urnovitz (2002) who has conducted his own research into Gulf War syndrome (GWS) and Mycoplasma (Urnovitz et al. 1999) is definite in his non-acceptance of the idea that systemic Mycoplasma infection is widespread.

> *My position on the role of Mycoplasma in CFS and GWS was stated under oath to the US Congress in January 2002: '…The Mycoplasma causal theory for GWS was based on poorly conducted research and the*

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claims had never been validated. Finally, an excellent controlled scientific experiment has put this matter to rest. In other words, I believe the controlled study [by Lo et al. 2000], using conventional clinical laboratory methods, has done an excellent job in suggesting that Mycoplasma plays little or no role in GWS.'

Why does one study using a well-established clinical laboratory method claim no role for this organism, while another research team claims GWS and CFS patients have 'systemic infections?' Urnovitz's main criticism of research which supports the Mycoplasma aetiology for FMS, etc., relates to the evidence apparently gained from polymerase chain reaction (PCR) tests :

'So what is the problem with the Mycoplasma papers? The abstracts of these studies seem to always claim that the patients are suffering from 'systemic infections.' If there were a truly systemic infection, where are the data showing the results of Mycoplasma cultures? Correlating PCR tests with microbial culture data is standard clinical laboratory practice. For example, a Mycoplasma/PCR study by Waring et al. (2001) can be obtained for free from the American Society of Microbiology web site. The authors correctly used the PCR technique as a pre-screen for culture. All that is published in the Mycoplasma PCR papers (that promote the Mycoplasma aetiology theory) are tables and charts claiming to show what percentage of patients is 'positive,' but never any correlative culture data. We cannot find any proper validation study comparing PCR and Mycoplasma culture data for any of the Mycoplasma species that some researchers claims are causing systemic infections in CFS and GWS patients. The only proper conclusion that can be drawn from these GWS/CFS studies is 'a large percentage (not even close to 100%) of CFS and GWS patients have genetically reactive samples, i.e., inconclusive laboratory results (Urnovitz 2002).

Urnovitz (2002) is also concerned at the damage that could be caused by the treatment protocol advised for attacking Mycoplasma infection. He expresses the problem cogently:

The argument is that, since the antibiotics can kill bugs like Mycoplasma, it must be the fact that Mycoplasma is being killed by the antibiotics that's making the patients feel better. Not only is this a circular argument, we're learning that the conventional wisdom that antibiotics work solely on microbes is inaccurate. The reasoning behind requiring manufacturers to describe an antibiotic's adverse side effects in package inserts is that these chemotherapeutic agents work on human genetic and protein material as well as microbial material. The number and severity of these adverse side effects is why regulatory agencies demand rigorous clinical trials on chemotherapeutic agents before they are allowed on the market. One cannot conclude that patients feel better on an antibiotic because it is killing Mycoplasma without a shred of clinical microbiological evidence. Our concern is that this unethical, off-label prescribing of antibiotic combinations will have significant adverse side effects on the patients taking them, as we have seen in the failure of anti-retrovirals prescribed to 'treat' HIV.

Comment

The analogy of flies swarming round a pile of garbage comes to mind when considering that unhealthy, possibly toxic, and/or nutritionally deficient, immune compromised, tissues might provide a fine environment for opportunistic organisms (viruses, fungi, Mycoplasmas, etc.). Can infectious agents cause conditions such as FMS? Or is it not more likely that the situation which allows the active presence of these organisms is associated with many of the underlying aetiological features of conditions such as FMS and CFS, and that any infectious organisms found to be present, while certainly adding to the adaptive burden, may not, in themselves, be causal?

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