

A possible etiologic agent; *Borrelia burgdorferi* in dilated cardiomyopathy*

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Borrelia burgdorferi (BB), a spirochete causing Lyme borreliosis, has been recently presented as an etiologic agent in dilated cardiomyopathy in Austria.

We examined 21 consecutive patients (aged 33-62, 12 men 9 women) with primary dilated cardiomyopathy. Four of them (19%) had antibodies to BB detected by the method "Dot immunobinding assay" which is an enzymatic serologic test. Twenty controls (aged 20-45, 12 men, 8 women) were also investigated and only one of them (5%) had antibodies to BB. All the other diseases which could give Borreliosis seropositivity, such as systemic lupus erythematosus, Infetious mononucleosis, syphilis etc, have been ruled out.

The important difference in seropositivity between the patients with dilated cardiomyopathy and controls (19% and 5%) gives further evidence that BB could be a possible etiologic agent in dilated cardiomyopathy in Turkey as well. [Turk J Med Res 1992, 10(3): 175-177]

Keywords: *Borrelia burgdorferi*, Dilated cardiomyopathy, Lyme disease

Lyme disease is a systemic disorder caused by the spirochete *Borrelia burgdorferi* (BB), transmitted by arthropods, especially by ticks.

The disease usually begins with a characteristic skin lesion (erythema chronicum migrans); neurologic, cardiac, and joint involvement may develop weeks to months later. Some manifestations last for years or even decades (1) (Table 1).

Even before the discovery of its etiologic agent, cardiac involvement has mainly been reported as transient, with early manifestations presenting especially as fluctuating conduction disorders, but also as acute myopericarditis and ventricular dysfunction (2,3).

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There is no report indicating that the long term effect of the disease is the reason for dilated cardiomyopathy until 1990. Since that time, only limited number of articles about this subject have been reported (1,4,5).

So, we wanted to study whether BB could be a possible etiologic agent in dilated cardiomyopathy in Turkey as well.

PATIENTS AND METHOD

We examined 21 consecutive patients (aged 33-62; 12 men 9 women) with primary dilated cardiomyopathy. Twenty controls (aged 20-45; 12 men, 8 women) were also investigated.

Sera from those with primary dilated cardiomyopathy and controls were frozen at -20°C until we tested. The serum samples were diluted 1:8. We used an enzymatic serologic test (Dot immunobinding assay-Detect a Dot Lyme test, Gull Lab. USA). IgM and/or IgG antibodies to BB were determined by this test (6).

Those with seropositive result could not recall a tick bite and any skin lesion.

Table 1.

LYME DISEASE	
Early manifestation:	
Skin lesion (Erythema chronicum migrans)	
Late manifestations:	
Musculoskeletal system :	Arthritis
Nervous system :	Lymphocytic meningitis, cranial neuritis, facial palsy, radiculoneuropathy, encephalomyelitis
Cardiovascular system :	Atrioventricular conduction defects myocarditis

RESULTS

Four of 21 patients (19%) with primary dilated cardiomyopathy had antibodies to BB detected by the method "Dot immunobinding assay". Only one of the controls (5%) had antibodies to BB (Table 2).

All the other diseases which could give false seropositivity, such as systemic lupus erythematosus, infectious mononucleosis and syphilis have been ruled out.

DISCUSSION

Lyme disease was originally recognized in Lyme, Connecticut, USA in 1975. Because of the varied clinical manifestations of this illness and the use of unstandardized serologic testing methods, diagnosis often uncertain and treatment outcomes are often difficult to evaluate.

The onset of the disease is usually heralded by the appearance of a pathognomonic skin lesion, known as erythema chronicum migrans. Unless antibiotic therapy is initiated early, the disease usually disseminates, often resulting in cardiac, neurologic or joint manifestations (3) (Table 1).

Definition of the disease is typically made by clinical evidence supported by serologic test results. A specific immune response against BB is usually detectable. IgM antibody generally first develops within 2 to 4 weeks after the onset of erythema chronicum migrans, peaks after 6 to 8 weeks of illness, and declines to the normal range after 4 to 6

months of illness in most patients. In some patients the IgM reappears antibody level remains elevated form many months or IgM antibody late in illness; these phenomena predict continued infection. The IgG antibody level is usually elevated within 6 to 8 weeks after the onset of the disease; peaks after 4 to 6 months of illness, and remains elevated indefinitely in patients with continued infection (3,6).

Cardiac manifestations of Lyme borreliosis were first reported by Steere et al. in 1980 (2), including conduction disorders, especially fluctuating degrees of atrioventricular block, but also transient cardiomegaly and left ventricular dysfunction.

Lyme carditis is uncommon, occurring in 8% of North American patients (3,7). The most common abnormalities are varying degrees of atrioventricular block causing palpitations, syncope, exertional dyspnea or lightheadedness or fatigue (7).

Transient ST-T abnormalities and reversible myocardial impairment are the other cardiovascular manifestations. Less commonly patients have had more diffuse cardiac involvement with reversible ventricular dysfunction and electrocardiographic changes suggestive of myopericarditis. But diffuse myocarditis may be a more frequent occurrence than clinical evidence alone would suggest. It is suggested that carditis is generally self-limited with complete recovery the rule (3).

Until 1990, there is no report indicating that the long term effect of the disease is the reason for dilated cardiomyopathy. A case, who had dilated cardiomyopathy for more than 2 years, documented BB in the myocardium (by transvenous endomyocardial biopsy), is reported by Gerold Stanek et al. from Austria (1).

In 1990 and 1991 Jutta Klein et al. from Austria examined the patients with dilated cardiomyopathy for antibodies to BB (Seropositive results were 29.6% and 33.3% respectively) (Table 2). In their patients treated with antibiotics, no clinical benefit was seen (4,5).

In our study 19% of patients and 5% of controls had antibodies to BB (Table 2). Because of the absence of data supporting the need for aggressive antibiotic therapy, we did not give any antibiotic drug.

All these findings suggest that BB may not only cause acute, but also chronic heart muscle disease and it could be a possible etiologic agent in dilated cardiomyopathy not only in USA or Europe, but in Turkey as well which is located between Asia and Europe.

Table 2.

	Patients	Controls
J. Klein (1990) (4)	81/24 (29.6%)	55/4 (7.3%)
J. Klein (1991) (5)	54/18(33.3%)	
G Pamir (1991)	21/4(19%)	20/1 (5%)

Dilate kardiyomyopatide Borrelia burgdorferi

Son yıllarda Avusturya'da, Lyme hastalığının etiyolojik ajanı olan *Borrelia burgdorferi* (BB) spiroketinin dilate kardiyomyopatiye sebep olabileceği ileri sürülmüştür.

Primer dilate kardiyomyopati tanısı alan 21 hasta (yaş 33-62, 12 erkek, 9 kadın) BB antikorları yönünden tetkik edildi. Enzimatik serolojik bir test olan "Dot immunobinding assay" metodu kullanıldı. Dört hastada (% 19) BB'ye karşı antikorlar tespit edildi. Buna karşılık kontrol grubunu oluşturan 20 kişinin (yaş 20-45, 12 erkek, 8 kadın) sadece birinde (%5) BB'ye karşı antikor gözlemlendi. Borreliosis seropozitivitesine sebep olabilecek, sistemik lupus eritematosus, infeksiyöz mononükleoz, sifilis gibi hastalıkların olmadığı gösterildi.

Dilate kardiyomyopatili hastalar ve kontrol grubu arasında Borreliosis seropozitivitesindeki önemli farklılık (%19 ve %5), BB'nin Türkiye'de de dilate kardiyomyopati etiyolojisinde araştırılması gereken bir faktör olduğunu ortaya koymuştur.

[Türk Tıp Araştırma 1992, 10(3): 175-177]

Anahtar Kelimeler: *Borrelia burgdorferi*, Dilate kardiyomyopati, Lyme hastalığı

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