Like most of the others, I was tested for all of the coinfections and came back negative with the exception of Mycoplasma Fermentans by PCR that I was tested for years ago. Never the less I am highly symptomatic of Babs, with symtoms including anorexic look and NOT sweating when appropriate and sweating at night.

I have NOT been treated specifically for Babs but am doing very well with nearly all symptoms abaiting. This pretty much is in stark contrast with what current treatment protocols say will happen. So if I have Babs, how is it that I am doing so well without specifically treating it?

It may have been by accident. I had pretty severe hypercoagulation/ISAC Syndrome and was treated with heparin quite some time before starting ABX for Mycoplasma and then Lyme. With my high state of remission I always wondered what happened, how did I get so well. Then I found this study, which is very new:

National Research Center for Protozoan Diseases, Obihiro University of Agriculture and Veterinary Medicine, Inada-cho, Obihiro, Hokkaido 080-8555, Japan.

We examined the inhibitory effects of three heparins on the growth of Babesia parasites. The multiplication of Babesia bovis, B. bigemina, B. equi, and B. caballi in in vitro cultures and that of B. microti in vivo were significantly inhibited in the presence of heparins, as determined by light microscopy. Treatment with various concentrations of heparin showed complete clearance of the intracellular parasites. Interestingly, a higher percentage of abnormally multidividing B. bovis parasites was observed in the presence of low concentrations of heparin. Furthermore, fluorescein isothiocyanate-labeled heparin was preferably found on the surfaces of extracellular merozoites, as detected by confocal laser scanning microscopy. These findings indicate that the heparin covers the surfaces of babesial merozoites and inhibits their subsequent invasion of erythrocytes.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=14693545&dopt=Abstract

Could this be the reason I have needed so few ABX? The majority of us are also dealing with hypercoagulation whether we realize if or not. Treating it could be as important as treating the coinfection, and you may be able to kill more bugs with the same stone.

Look for the Cure/Hypercoagulation

Forwarded Message:

Subj:	**Babesia / Disseminated intravascula coagulation**
Date:	7/2/2006 4:07:23 PM Pacific Standard Time
From:	Gjgilberg
To:	Neural T

Comparative pathogenesis of human WA1 and Babesia microti isolates in a Syrian hamster model.

Wozniak EJ, Lowenstine LJ, Hemmer R, Robinson T, Conrad PA.

Department of Pathology, Immunology, and Microbiology, School of Veterinary Medicine, University of California, Davis 95616, USA.

The pathogenesis of a newly recognized, molecularly and antigenically distinct human babesial isolate (WA1) and Babesia microti, the common cause of human babesiosis in the United States, were compared in a Syrian hamster model.

A group of 33 adult female hamsters were inoculated intraperitoneally with either WA1-infected, B. microti-infected, or uninfected hamster erythrocytes.

All WA1-infected animals became parasitemic by postinoculation (PI) day 3 or 4 and were severely lethargic and dyspneic by PI days 6 to 10. Death often occurred spontaneously by PI day 10, with parasitemia of 12 to 90%.

Hamsters inoculated with B. microti became parasitemic by PI day 7 and developed peak parasitemia (42 to 60%) by PI day 14 that subsequently decreased to low or undetectable values. Although the B. microti-infected hamsters developed severe anemia, they generally remained asymptomatic.

Postmortem examination of WA1-infected hamsters revealed intravascular aggregates of large mononuclear inflammatory cells that occasionally occluded small to medium veins, pulmonary leukoclastic phlebitis, thrombosis, and multifocal coagulative necrosis in the heart, spleen, lung, and liver.

No vascular lesions or areas of coagulative necrosis were detected in any B. microti-infected or control hamsters.

The results of this study suggest that marked leukocytosis followed by acute necrotizing phlebitis resulting in **disseminated intravascular coagulation,** thromboembolism, and infarction may be central to the pathogenesis of WA1 infections.

PMID: 8905583 [PubMed - indexed for MEDLINE]