BACILLARY ANGIOMATOSIS
AND OTHER BARTONELLA INFECTIONS
(SUMMARY)

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Until seven years ago, members of the Bartonella and Rochalimaea genera were not viewed as global pathogens, but rather as important causes of regional disease localized to discrete regions of South and Central America, Eastern Europe, and the Soviet Union. Only two disease-associated species were recognized: B. bacilliformis and R. quintana. Today, B. quintana (formerly R. quintana) and a number of previously-uncharacterized species within the unified genus, Bartonella, are commonly-recognized pathogens in large parts of the world and are associated with a wide spectrum of pathology. A number of factors may explain this new perspective, including the development of alternative, more sensitive, non-culture-based methods of microbial identification. In 1990, Bartonella henselae was first identified using a consensus rDNA PCR approach, and was associated with a novel form of angioproliferative pathology: bacillary angiomatosis (BA). We now recognize both B. henselae and B. quintana as causes of BA. B. henselae is also responsible for most cases of cat scratch disease. More than ten Bartonella species and sequence variants are currently recognized. With the development of cultivation, in situ techniques, and methods for genetic manipulation, our understanding of these organisms has expanded dramatically.

Bartonella infections are usually asymptomatic in the natural host, despite the proclivity of these organisms for the intravascular compartment and their tendency to cause sustained bacteremia. A wide variety of domestic and wild animals serve as reservoirs for Bartonella species; humans may serve as a reservoir for B. quintana. Some bartonellae express factors that mediate tight adherence with, and deformation of erythrocyte membranes. In humans, disease is manifest by a granulomatous tissue response (e.g. cat scratch disease) in most immunocompetent host, and by an angioproliferative response (BA, bacillary peliosis –BP, or verruga peruana– BP). BA and BP are usually seen in immunocompromised hosts. VP occurs in the Andean regions of South America as a response to chronic B. bacilliformis infection.

Descriptions of angioproliferative pathology other than VP (BA and BP) first appeared in the 1980's, suggesting that either BA and BP were previously ignored, or that susceptible hosts prone to, and/or competent B. henselae and B. quintana strains capable of, BA and BP are relatively new developments. Mechanisms of Bartonella associated angiogenesis are unclear; however, all three species associated with this host response may share similar virulence factors. A directly-acting bacterial angioproliferative factor, or a more indirect mechanism such as stimulation of host cytokine expression and angiogenic pathways, are both possibilities.
Bartonella infections, caused by currently-recognized and not-yet-recognized species, are likely to remain important health problems for the foreseeable future. Some of the important unanswered questions concern modes of Bartonella transmission to humans, modification of Bartonella virulence by arthropod vectors, the population structure of disease-associated Bartonella strains, mechanisms of angioproliferation and of bacterial translocation to the bloodstream, and the host factors that predispose towards one form of pathology versus another.

REFERENCES


