Differentiating Relapse from Same-Strain Reinfection in Recurring Gram-Negative Bacteremia

Sir—Pulsed-field gel electrophoresis of macrorestricted genomic DNA, the molecular strain typing method used by Wendt et al. [1, 2] to classify isolates recovered during recurrent bacteremia as “same” versus “different” in comparison with a patient’s previous blood isolate, is a powerful tool that is well suited to this purpose. However, no molecular typing method can define the mechanism responsible for the second appearance of a given strain in an individual patient, which instead becomes a matter of judgment that should be guided by epidemiological data and surveillance cultures.

Although same-strain recurrences can be due to persistence of the organism within the host (i.e., a true “relapse” as assumed by Wendt et al. [1, 2]), reintroduction of the same strain from a persisting external reservoir, whether in the environment or in the patient’s own bacterial flora, is also possible. This latter scenario constitutes “same-strain reinfection” and is indistinguishable by molecular techniques from true relapse. Early same-strain reinfection is probably the most common cause of what masquerades as relapse in women who receive single-dose antimicrobial therapy for uncomplicated cystitis. In such cases, the initial urinary pathogen commonly persists in the vagina and rectum, despite its disappearance from the urine, and can reenter the urinary tract once antimicrobial activity there has waned [3].

The distinction between true relapse and same-strain reinfection is of more than academic importance, since different interventions are required for each condition. Intensified therapy designed to eliminate a persisting internal focus of infection will be of little value if it fails to address a persisting external reservoir that is the true cause of same-strain recurrence. Infection control efforts may be highly relevant for same-strain recurrences that involve a persisting reservoir outside the host, whereas they would be irrelevant for true relapses from an internal focus.

Wendt et al. [1, 2] noted that retained vascular catheters were a unifying theme among their subjects with presumed relapsing gram-negative bacteremia. This clinical circumstance indeed would argue for relapse rather than same-strain reinfection as the mechanism for the observed same-strain recurrences. However, it should be recognized that this probability remains an inference based on epidemiological data, not a direct conclusion from molecular strain typing.

James R. Johnson
Infectious Diseases Section, Minneapolis Veterans Affairs Medical Center, and Department of Medicine, University of Minnesota, Minneapolis, Minnesota

References

Thrombocytopenia and Borrelia burgdorferi: An Association Remains Unproven

Sir—In their report in the April issue of Clinical Infectious Diseases of a patient with autoimmune thrombocytopenia, Stefan et al. [1] propose that the illness was triggered by a possible Borrelia burgdorferi infection 18 months earlier. The authors claim, on the basis of 2 publications, that an association exists between B. burgdorferi and thrombocytopenia. First, they cite a previous report in this journal of a patient for whom this association was suggested [2]. However, the authors fail to indicate that in a subsequent letter evidence was provided that the patient was coinfected with the agent of human granulocytic ehrlichiosis (HGE), an infection closely associated with thrombocytopenia [3]. Second, they cite a report of 6 patients with thrombocytopenia and clinical or serological evidence of B. burgdorferi infection [4]. However, this report from Westchester County, New York, was published prior to the recognition of HGE in this area. Consequently, specific assays for detection of HGE were not available. The omission is critical, because coinfection with HGE is not rare in this geographic area [5] and because false-positive serological reactivity to B. burgdorferi may occur in patients with HGE [6].

Stefan et al. [1] have not provided any new convincing evidence to associate B. burgdorferi infection with thrombocytopenia. Not only was their patient’s rash undocumented, but the laboratory data provided did not establish B. burgdorferi infection. The presence of 2 bands on IgG immunoblot is non-diagnostic. Even if the patient did have Lyme borreliosis at some point between the last normal platelet count (2 years before) and the development of thrombocytopenia, there is no reason to assume an etiologic relationship. A more plausible diagnosis for the patient in the report of Stefan et al. is idiopathic thrombocytopenic purpura.