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Reinfection Versus Relapse in Urinary Tract Infection

Sir—The much-needed trial by Dow et al. [1] regarding treatment duration for urinary tract infection (UTI) in patients with spinal cord injury and the accompanying editorial commentary [2] raise questions regarding the determination of whether isolates from recurrent (posttherapy) episodes of UTI represent relapse or reinfection and determination of the possible sources of such infections. First, although various methods can be used to classify isolates from recurrent episodes of UTI as the same strain or a different strain than the pretherapy isolate, even isolates of the same strain can represent reinfection if there is a persisting external reservoir from which the organism can be reintroduced into the host’s urinary tract. Thus, whereas isolates of different strains almost certainly represent reinfection, isolates of the same strain are ambiguous with respect to whether they represent reinfection or relapse. This ambiguity can lead to overestimation of relapse rates.

Second, because of the relatively high prevalence of Klebsiella species, Enterococcus species, and Escherichia coli among pretherapy isolates from urine in the study of Dow et al. [1], an inference that a posttherapy isolate of the same species represents the same strain as the pretherapy isolate may be erroneous. Subspecies typing methods, such as PFGE, are needed here. It is unclear why Dow et al. [1] reserved PFGE analysis for only selected same-species posttherapy isolates. Third, even PFGE analysis may not provide unambiguous results. For example, it is statistically improbable that a patient whose pretherapy urine sample yielded Acinetobacter anitratus would have recurrent UTI due to an unrelated strain of A. anitratus, given that the overall prevalence of infection by Acinetobacter species in the study population was 10%, and only a subset of those infections, presumably, was due to A. anitratus [1]. It is perhaps as likely that the PFGE results in this instance were in error, or that the strain, while residing in the patient (or in a patient-associated reservoir), underwent genetic rearrangements that produced the observed PFGE profile alterations, which led to a false assessment that this was a different strain from the pretherapy isolate.

Finally, 70% of the study subjects were men [1]. Because the prostate gland is a common source for relapsing UTI in men [3] and is usually involved in cases of febrile UTI in men, despite the absence of localizing symptoms [4], it may be that some of the relapses observed by Dow et al. [1] derived from a persistently infected prostate rather than the upper urinary tract, as was proposed. The tenacity of prostatic infection [3] would be consistent with the superior microbiological efficacy of the longer treatment course that was observed by Dow et al. [1].

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Reply to Johnson

Sir—We thank Dr. Johnson [1] for his interest in and comments on our study. To determine outcomes of relapse or reinfection we used definitions commonly applied in clinical trials of urinary tract infection (UTI). We agree that isolation of the same species after therapy may possibly result from reinfection with the same strain from an external source. This has been well documented for young women with acute uncomplicated UTI [2], although similar observations for complicated UTI have not been reported, to our knowledge. Despite this, reinfection rates identified with the study definitions were similar for both treatment arms, whereas relapse occurred only in the 3-day treatment arm. Surveillance cultures of sites of colonization, the likely source of same-strain reinfection, did not reveal persistent colonization in either arm after treatment. These observations support the conclusion that outcomes classified as relapse did represent relapse rather than reinfection. PFGE typing was only performed for organisms associated with late relapse. The expectation was that relapse would usually be identified early after therapy, so late relapse, of which there were only 2 occurrences, would more likely represent reinfection. The majority of study subjects were inpatients on the spinal cord injury unit and exposed to nosocomial pathogens, and Acinetobacter species are well recognized causal organisms in this situation. Thus, reinfection with Acinetobacter
anitratus is certainly “statistically probable” [3]. Three Acinetobacter isolates from the patient from our study [4] with late relapse—1 isolated 2 days before initiation of antimicrobial therapy, 1 immediately before therapy, and another at week 6 of therapy—were analyzed by PFGE. The 2 pretherapy isolates were similar, but the third isolate was clearly distinct, using standard criteria for interpretation. Although we cannot comment on the suggestion of genetic reassortment, PFGE typing of Acinetobacter species has been widely used in epidemiologic studies and is considered a valid method [3].

The prostate gland is certainly a potential source for relapsing UTI in men. There would be no specific way to identify this localization. The study by Ulleryd et al. [5] describes febrile UTI, so it is not applicable to our study population. As noted in the article, a previous study of UTI localization in a population of men with spinal cord injuries reported that almost 50% of subjects with bacteriuria had occult upper urinary tract involvement [6]. In fact, a 2-week course of therapy is likely not sufficient to eradicate prostatic bacteriuria, so the absence of relapses in the 14-day group suggests that the prostate was not a common source of infection.

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