Urinary Tract Infection Caused by Enterobacter taylorae

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We present a case of urinary tract infection caused by Enterobacter taylorae in a 70-year-old male with renal lithiasis. The microorganism was isolated in significant numbers from a urine culture. The disappearance of clinical symptoms after antibiotic treatment points to the participation of this microorganism as the etiological agent in the infection.

In 1985, Farmer et al. (2) proposed that the bacterium previously included in Enteric Group 19 should be recognized as a new species in the family Enterobacteriaceae named Enterobacter taylorae. Farmer et al. also reviewed the new species and biogroups of Enterobacteriaceae (1), including 31 strains of E. taylorae isolated from human sources. Of these, only one (3.2%) was obtained from urine, the most common origins being wounds (45%) and feces (16%). It seems, therefore, that the isolation of E. taylorae from the urine of patients with urinary tract infection is not a common occurrence. This fact prompted us to report the present case, in which this microorganism was clearly implicated as the etiological agent.

A 70-year-old man with a 10-year history of repeated urinary lithiasis was treated in the emergency service for disuria and hematuria. Urinalysis showed a pathological sediment with more than 25 leukocytes per field, pyuria, abundant erythrocytes, and bacteriuria. A sample of urine was taken for bacteriological culture, and treatment with trimethoprim-sulfamethoxazole was begun. The sample was plated by using a quantitative method with a calibrated loop (0.01 ml) onto cystine lactose electrolyte deficient (CLED) agar and MacConkey agar, which were incubated at 37°C for 18 to 24 h. More than 100,000 CFU of a gram-negative, lactose-positive bacillus per ml was obtained. The biochemical identification was performed with the GNI card (Vitek System Co., St. Louis, Mo.), which identified the microorganism as E. taylorae (biotype number 3030700373) with 99% certainty; the final identification was confirmed by conventional tests (2). Susceptibility studies with the GNS card (Vitek) showed the microorganism to be resistant to aztreonam (MIC, >32 μg/ml), carbenicillin (MIC, 64 μg/ml), cefazidime (MIC, >32 μg/ml), ceftriaxone (MIC, 64 μg/ml), cefuroxime (MIC, >32 μg/ml), mezlocillin (MIC, >256 μg/ml), piperacillin (MIC, >128 μg/ml), ampicillin (MIC, >32 μg/ml), cefazolin (MIC, >32 μg/ml), and cefoxitin (MIC, >32 μg/ml) and susceptible to chloramphenicol (MIC, 8 μg/ml), ciprofloxacin (MIC, <0.5 μg/ml), imipenem (MIC, <4 μg/ml), tobramycin (MIC, <0.5 μg/ml), amikacin (MIC, <2 μg/ml), cefotaxime (MIC, 8 μg/ml), gentamicin (MIC, <0.5 μg/ml), ticarcillin (MIC, 32 μg/ml), and trimethoprim-sulfamethoxazole (MIC, <10 μg/ml).

Since most of the E. taylorae strains isolated to date have originated in organic sites which are not generally sterile (feces, respiratory tract, cutaneous wounds), it is difficult to evaluate the true clinical significance of these isolates. Nevertheless, the isolation of five strains from blood and one from cerebrospinal fluid suggests a pathogenic capacity (2). In addition, Westblom and Coggins recently reported the first case of osteomyelitis caused by E. taylorae in a previously healthy patient, demonstrating the implication of this microorganism in abscess formation (4).

In our case, E. taylorae was isolated in pure culture, with a urinary count considered to be significant, and in the presence of symptoms of urinary infection and a satisfactory response to antibiotic treatment, all of which appears to indicate the direct participation of this microorganism in the infectious process. The patient being male, it is difficult to establish the source of the organism (portal of entry) since, in spite of the fact that E. taylorae has been isolated in the feces of healthy persons, it was impossible to demonstrate enteric contamination in this case. In patients with renal lithiasis, it is common to find colonization by different microorganisms which seldom produce true urinary infections (3). The most likely portal of entry for E. taylorae is, therefore, previous urethral contamination of an environmental origin, with access to the urinary system by the ascending route.

Attention must be drawn in this case to the association between E. taylorae and renal lithiasis, largely because the microorganism is not a urease producer, a fact which prohibits a direct contribution to the process of bacterial lithogenesis. This episode should probably be considered one of the multiple urinary infections which habitually occur in patients with chronic lithiasis and which are not always related to the ureolytic activity of the etiological agent.

LITERATURE CITED


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