Concurrent Fecal Colonization with Extraintestinal Pathogenic Escherichia coli in a Homosexual Man with Recurrent Urinary Tract Infection and in His Male Sex Partner

James R. Johnson and Parissa Delavari

Medical Service, Veterans Affairs Medical Center, and Department of Medicine, University of Minnesota, Minneapolis

A 72-year-old homosexual man experienced frequent recurrences of symptomatic urinary tract infection (UTI) putatively due to the same strain of extraintestinal pathogenic Escherichia coli, despite receiving repeated courses of seemingly appropriate antibiotic therapy. Both the patient and his male sex partner were found to have fecal colonization with the strain recovered from the patient’s urine, which exhibited characteristics of the E. coli O1/O2:K1:H7 clonal group (which is associated with urosepsis), whereas genetically distinct E. coli strains were recovered from a jar of petroleum jelly used by the couple during sexual activity.

Escherichia coli is the most common cause of urinary tract infection (UTI). The immediate source of the causative strain is thought usually to be the host’s own fecal and/or vaginal flora [1]. How individuals come to be colonized with the distinct extraintestinal pathogenic E. coli (ExPEC) strains that cause most UTIs is unknown.

Direct person-to-person transmission of ExPEC through sexual contact has been inferred from several epidemiological observations. Among heterosexuals, these observations include the sequential or simultaneous occurrence of UTI episodes due to the same strain in both members of sexually active couples [2–4] and colonization with the strain responsible for the woman’s UTI in male partners of women with acute cystitis [5–8]. Among homosexual men, the occurrence of acute UTIs due to E. coli strains that exhibit characteristics of ExPEC has suggested the possibility of urethral inoculation of pathogens from the partner’s fecal flora via anal intercourse [9]. However, concurrent fecal colonization of a patient with acute UTI and of his male sex partner with the strain of E. coli recovered from the patient’s urine has not been demonstrated. We provide evidence of this phenomenon discovered during a search for a possible external reservoir for a strain of E. coli responsible for recurrent UTI in a sexually active homosexual man.

Methods. Urinalysis and urine cultures were performed by the Minneapolis Veterans Affairs Medical Center clinical laboratory. Susceptibility of the isolated strain to 21 antibiotics was determined by broth microdilution. Fecal swabs were collected by the subjects from soiled toilet tissue [5] and used to inoculate nonselective broth, which, after overnight incubation, was streaked on MacConkey’s agar. Bacilli were defined as E. coli if they were lactose positive, indole positive, and gram negative and exhibited colonial morphology consistent with E. coli [5]. Three colonies of putative E. coli from each culture plate were arbitrarily selected for further analysis. (This approach yields a 97% probability of including the predominant fecal strain [10].) A jar of petroleum jelly from the patient’s household was swabbed over its entire surface, and the swab was then processed as the fecal swabs had been. All available colonies of putative E. coli were analyzed further.

E. coli colonies were subjected to randomly amplified polymorphic DNA (RAPD) analysis with use of 3 separate primers (i.e., primers 1247, 1254, and 1281) [11]. If indistinguishable with use of all 3 primers, isolates were defined as being of the same strain. Detection of 32 virulence-associated factors characteristic of ExPEC and the 12 alleles of papA (P fimbrial structural subunit gene) was done using established PCR-based methods [11, 12].

This work was performed in accordance with the ethical standards of the relevant local investigative review boards and with the Helsinki Declaration of 1975, as revised in 1983.

Results. The patient was a 72-year-old man with type 2 diabetes mellitus, panhypopituitarism, hypertension, hyperlipidemia, depression, coronary artery disease, benign prostatic hyperplasia, obstructive sleep apnea, and erectile dysfunction. Implantation of a penile prosthesis had been delayed because of new-onset recurrent UTIs.

Each UTI episode manifested as rapidly progressive dysuria.
Figure 1. Randomly amplified polymorphic DNA (RAPD) analysis of fecal and urinary Escherichia coli isolates. The same 5 isolates (A–E) were analyzed using RAPD primers 1247 (lanes 2–6), 1254 (lanes 8–12), and 1281 (lanes 14–18). Isolate A is from the patient’s feces, isolate B is from the partner’s feces, and isolates C, D, and E are from the patient’s urine. Lanes 1, 7, 13, and 19, 100-bp ladder. The 2 additional fecal isolates were obtained from each of the 2 subjects and analyzed using primers 1247 and 1254; they yielded RAPD profiles that were indistinguishable from those shown for isolates A and B, respectively, whereas the 7 isolates from the jar of petroleum jelly collectively yielded 2 unrelated profiles (not shown).

and frequency and urgency of urination; flank, pelvic, and perineal pain were absent. Only the second episode had been accompanied by fatigue, fever, chills, and slight leukocytosis (WBC count, 13,300 cells/mm³; differential count, normal). Findings of physical examinations (including prostate examination) were always normal. Urine samples obtained before the initiation of antimicrobial therapy consistently revealed >15 WBCs per high-power field (hpf) on microscopy, and, on culture, they yielded >10⁵ cfu/mL of E. coli that was susceptible to all antibiotics tested. Symptoms always resolved promptly after initiation of therapy with trimethoprim-sulfamethoxazole (1 double-strength tablet twice daily), only to return within 2 weeks after completion of therapy. The durations of treatment were 10 days, for episodes 1 and 2; 14 days, for episode 3; and 28 days, for episodes 4 and 5.

The patient had no history of previous UTIs or sexually transmitted diseases. He had sex several times weekly with his monogamous, cohabiting male partner. The patient’s erectile function was insufficient for insertive anal intercourse, but the couple practiced mutual oral sex, mutual masturbation with the use of petroleum jelly lubricant, and receptive anal intercourse (partner to patient) using the same lubricant.

Urological evaluation revealed nothing significant. Bladder ultrasonography showed no post-void residual urine. Excretory urography showed only medial deviation of the left ureter. Abdominal CT confirmed this finding and revealed no mass or other abnormalities. Cystourethroscopy showed only prostatic hypertrophy and bladder trabeculations.

When the patient was examined in the infectious diseases clinic of our institution after his fifth symptomatic UTI episode in 4 months, he had recently completed a course of trimethoprim-sulfamethoxazole therapy and was asymptomatic. Findings of a genital examination were normal. Urine samples contained 17 WBCs/hpf and <10⁴ cfu/mL of susceptible E. coli. Levofloxacin was prescribed (500 mg po q.d. for 28 days), to be taken in case of recurrent symptoms. Arrangements were made for cultures of swabs of the couple’s feces and the jar of petroleum jelly.

Three weeks later, the patient, still asymptomatic, returned for another examination, bringing with him swab samples for culture. A urine sample now contained 40 WBCs/hpf and >10⁵ cfu/mL of susceptible E. coli. Five days later, UTI symptoms returned and levofloxacin therapy was begun. Symptoms resolved promptly. A new urine sample showed 11 WBCs/hpf but was sterile. Levofloxacin therapy was continued for a total of 28 days. In the subsequent 2 years, the patient has had no further symptomatic episodes, and multiple urine cultures have been sterile.

RAPD analysis was performed on 3 colonies of E. coli from each of the fecal cultures done for the patient and the patient’s partner and from the patient’s pretherapy urine culture and on all 7 E. coli colonies from the jar of petroleum jelly. The fecal and urinary colonies exhibited a homogeneous profile with use of each of 3 RAPD primers (figure 1); this was molecular evidence that, at the time that samples were obtained, the predominant fecal strain in both subjects was the patient’s urinary strain. In contrast, the E. coli colonies from the jar of petroleum jelly had 2 unrelated RAPD profiles (not shown).

Extended virulence genotyping revealed that the patient’s urinary isolate and both subjects’ fecal isolates exhibited a ho-
mogeneous profile of virulence-associated factors that was characteristic of the O1/O2:K1:H7 clonal group [12]. The factors present included the following: pap elements (encoding P fimbriae), papA allele F11 (a structural subunit gene), papC allele II (the adhesin gene), fimH (the type 1 fimbriae gene), iutA (the aerobactin receptor gene), fyuA (the yersiniabactin receptor gene), iraN (a siderophore receptor gene), kpsMT II variant K1 (the K1 capsule gene), flic variant H7 (the A7 flagellin gene), evaC (the colicin V gene), traT (a gene associated with serum resistance), iss (a gene associated with serum survival), and malX (a pathogenicity island marker).

Discussion. We describe concurrent fecal colonization of a patient who had recurrent UTI and of his male sex partner, with the strain of E. coli recovered from the patient’s urine. An inanimate object used by the couple during sexual activity yielded only E. coli strains with unrelated RAPD profiles. The recurrent episodes of UTI were seemingly terminated by an extended course of levofloxacin therapy.

This is the first available evidence of fecal colonization of both members of a sexually active homosexual couple with a strain of E. coli that caused UTI in one of the partners. The strain exhibited virulence characteristics consistent with the O1/O2:K1:H7 clonal group, which is a prominent cause of urosepsis [12]. A similar colonization pattern was previously described in a heterosexual couple in which the man was fecally colonized with the same pap-positive E. coli strain as that which colonized the woman’s intestine and vagina and had caused recurrent episodes of pyelonephritis [5].

Sexual transmission of ExPEC between homosexual men who practice anal intercourse has been postulated [9] but not previously demonstrated. Whether, in the present case, the shared strain was actually transmitted between the patient and his partner is unknown, but sexual transmission seems quite plausible, considering the couple’s frequent oral and anal sexual contact. However, even if sexual transmission did occur, it is intuitively less likely that the patient’s multiple recurrent episodes of UTI were due to repeated reintroduction of the strain from his partner’s or his own feces than that the recurrent episodes were due to relapse from a persisting endogenous focus within the prostate or, possibly, the bladder. Resolution of the UTI episodes after a prolonged course of treatment with a prostate-penetrating antibiotic is consistent with the existence of a prostatic reservoir [13]. Also, at a mechanistic level, it is less apparent in this case than it would be in the case of men who engage in insertive anal intercourse how the patient, who had insufficient erectile function for penetration, could have repeatedly reintroduced a strain from his partner’s fecal flora into his own urinary tract. Additional molecular surveillance testing of the couple after the completion of antibiotic therapy might have helped clarify whether the partner harbored a persisting reservoir of the strain of E. coli that caused infection in the patient or was only transiently colonized from a primary reservoir in the patient. Of note, although the jar of petroleum jelly was considered a candidate reservoir that could have re-inoculated the patient’s urethra during masturbation, molecular typing revealed that it contained only E. coli strains with unrelated RAPD profiles.

The inference that the multiple positive urine cultures (n = 7) for the patient yielded the same strain of E. coli is based primarily on the uniform susceptibility profile of the isolates. Since only 59% of urinary E. coli isolates from outpatients at the Minneapolis Veterans Affairs Medical Center are fully susceptible (unpublished data), the probability that 7 such isolates would exhibit this profile by chance alone is only 0.595 or 0.02.

The present case demonstrates the occurrence of fecal colonization with the same strain of E. coli in male homosexual partners, and, although it does not definitively confirm homosexual transmission of the E. coli strain responsible for the UTI, it illustrates that molecular surveillance for this phenomenon is feasible in a clinical setting. Person-to-person transmission of ExPEC during sexual activity would seem as likely among homosexuals [9] as among heterosexuals [2–8]. Whether, and how frequently, such transmission leads to symptomatic UTI in one or both partners may be worth defining because of its potential relevance to UTI prevention.

Acknowledgments

Francine Lebahn assisted with subject enrollment and institutional review board approvals. Dave Prentiss prepared the figure. Ann Emery helped with manuscript preparation.

References