Transmission of Uropathogens between Sex Partners


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Epidemiologic evidence and several case reports suggest that Escherichia coli causing urinary tract infection (UTI) may be transmitted between sex partners. In order to test this hypothesis, urinary, vaginal, and fecal E. coli isolates from 19 women with UTI were compared with E. coli found in random initial voids from their most recent male sex partner. E. coli was isolated from 4 of 19 male sex partners. In each case, the E. coli isolated from the man was identical by pulsed-field gel electrophoresis and bacterial virulence profile to the urinary E. coli from his sex partner.

Sample collection. Clean-voided urine specimens were obtained from UTI patients and cultured, and E. coli was identified often recurring in infection thought to be caused by ascendance of bowel flora into the bladder [1]. Frequent sexual intercourse [2] may cause trauma as well as move vaginal or bowel bacteria (or both) to the urethral opening, facilitating ascent to the bladder [1]. Sexual activity may also transmit uropathogens. First UTI diagnoses rapidly increase around the age of first sexual intercourse [3]. Using data from a cross-sectional survey among college students and methods described previously [4], we estimated that initiating sexual activity of any type increases UTI risk 3.5-fold (95% confidence interval: 2.5–5.1).

In order to test our hypothesis of sexual transmission of UTI, we used pulsed-field gel electrophoresis (PFGE) to compare Escherichia coli causing UTI in 19 sexually active women to the E. coli isolated from random initial stream urine specimens from their most recent male sex partner.

Materials and Methods

Study protocol. We enrolled consenting women aged 18–40 who presented to the University of Michigan Health Service during fall 1995 with a UTI diagnosis, two or more urinary symptoms, and a urine culture positive for E. coli; who had had recent vaginal intercourse; who were not pregnant, diabetic, or recently catheterized or hospitalized; and whose most recent male sex partner participated. Men enrolled within 4 days of the woman. Participants independently completed a self-administered questionnaire regarding medical and sexual history. Couples were compensated ($50).

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Informed consent was obtained from all study participants. The study protocol was approved by the Institutional Review Board at the University of Michigan School of Public Health.

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in a subclone pRP7 to identify CapIII gene clusters [8]. Isolates positive for prf by dot blot were analyzed for P fimbral adhesin subtypes papGJ96, papGIA2, and prsGJ96, using type-specific DNA probes. The probes for papGJ96, papGIA2, and prsGJ96 were isolated directly by PCR using primer pairs (5'-CTGTCAGGCTGTAATGATGC-3' and 5'-CAGGATAGAAACATATACGGGCA-3'; 5'-GGAGACGTTAACTCTTATCAGG-3' and 5'-CCAAATACGTCGAAAATGACG-3'; 5'-CTGTCAGGCTGTAATGATGC-3' and 5'-AACTCTGGCGCGTACAACAG-3', respectively) derived from GenBank sequences (accession nos. M20146, M20181, and X61238) and total DNA from strains J96 (papGJ96 and prsGJ96) and C1212 (papGIA2). We amplified (30 cycles: 95°C, 1 min; 60°C, 30 s; 74°C, 30 s) using a Microcycler thermal cycler (Eppendorf, Fremont, CA).

PFGE. Purification, rare-cutter restriction, and PFGE of minimally sheared E. coli DNAs were performed as previously described [5].

Data analysis. We used Filemaker Pro and Microsoft Excel for data entry and SAS for data management and analysis.

Results

We invited 149 women to participate. Of these, 86 (58%) refused: 37 said their partner was out of town, 23 said their partner would not participate, and 26 simply refused. Twenty-eight of the 63 consenting women had a clinically diagnosed E. coli UTI and met other eligibility criteria. Fifteen of 19 women had >100,000 cfu of E. coli/mL of urine. Sex partners of 21 of 28 women participated. Two pairs were excluded because of the date of last sexual contact of the male was prior to that reported by the female, leaving 19 couples. Female participants were younger than their partners (mean, 22.4 years [SD, 4.6] vs. 24.8 [SD, 6.1]), and 32% reported a UTI history.

Four (21%) men had E. coli in their urine (95% confidence interval, 3%–38%); colony counts ranged from 2000 to 10,000 cfu/mL. Four men reported urinary symptoms: 2 of 4 colonized men reported urgency, 1 of 15 uncolonized men reported painful urination, and another uncolonized man reported chills and back pain. No man sought treatment for symptoms at the Health Service.

By virulence profile and PFGE, the E. coli in the colonized men was identical to the E. coli found in their sex partner’s urinary, vaginal, and/or rectal flora (see figure 1). Lanes B2 and B4 have a high-molecular-weight band missing in B1 and B3, which is substantially less intense than all other bands. This may represent partial-digestion products or possibly a plasmid. Lane C1 has a similar band missing in C2 and C3. One woman with a colonized partner did not have E. coli in her vagina; another had an inadequate fecal specimen.

Most women (15/19) carried E. coli in their vaginas, and all 18 with adequate fecal specimens carried E. coli in their rectums. In 11 cases, bladder, vaginal, and rectal E. coli had identical virulence profiles. By virulence profile, there were 4 cases in which the uropathogenic E. coli was isolated from the rectum but not the vagina and 2 in which the uropathogenic E. coli was isolated from the vagina but not the rectum. Among cases with adequate fecal specimens, there was only 1 in which we failed to isolate a uropathogen from either the vagina or rectum.

Each UTI isolate was distinct by PFGE or plasmid profile or both. On the basis of 12 bacterial virulence factors, we observed 12 unique virulence profiles; profile was not associated with co-colonization, although 2 of 4 co-colonized couples were colonized with E. coli positive only for fim.

We used previously published data to determine the probability that we would observe co-colonization of couples by chance alone. By virulence profile and Southern blot analysis [7], we previously identified a minimum of 125 distinct E. coli uropathogens. The probability that in 19 samples we would select 19 different isolates from 125 possible is .24; thus, 125 is probably a conservative estimate. The frequency of each of the 125 strains ranged from .005 to .06 [7]. Under the conservative assumptions that there are only 125 E. coli uropathogens, and that the probability of male colonization is the observed 4/19, the probability that a man would be colonized with the same strain as his sex partner by chance alone ranges from .001 (.005 × 4/19) to .013 (.06 × 4/19) if all strains are equally likely to be transmitted. Assuming the samples in the current study come from the distribution previously observed in the
125 strains [7], and that co-colonization occurred in 4 couples, the probability of observing co-colonization with the identical organism all four times by chance alone is .00000006.

While the numbers are extremely small, co-colonized partners had more lifetime sex partners (for women, a mean of 13.0 [SD, 14.5] vs. 4.6 [SD, 6.0] for non–co-colonized; for men, a mean of 6.8 [SD, 3.4] vs. 5.3 [SD, 7.1] for non–co-colonized) and partnerships of shorter duration (mean, 332 days [SD 518] vs. 537 [SD, 495]) than noncolonized partners. Colonized men had engaged in sex with their study partner more recently (1.5 vs. 2.8 days, \( P = .13 \)) but presented for culture only slightly sooner than noncolonized men (1.5 vs. 1.9 days, \( P = .70 \)). There was little or no difference in frequency or type of sexual behaviors, condom use or other birth control method, UTI history, or other behavioral variables. No couples engaged in anal intercourse. All male participants were circumcised.

No symptomatic male sought medical attention at the Health Service in the 3 months following enrollment. The 3-month recurrence rates were higher among women with co-colonized than noncolonized partners (3/4 vs. 6/15).

Discussion

Sex partners of women with UTIs are colonized with apparently identical uropathogens far more often than would be expected by chance. As most of the 6 million women presenting annually with UTI in the United States do not have recurring UTI (\( \geq 3 \) during a 12-month period) [1], the possibility that even 20% of these infections might be avoided by adopting preventive behaviors could significantly impact morbidity and treatment costs. This preliminary study suggests that uropathogens may be transmitted directly by sexual contact.

Three single case reports suggest sexual transmission of UTI from female to male [9–11] and one from male to female [12]. Among servicemen in the British Army, married men accompanied by their wives compared to unaccompanied men had a 11.9-fold increased risk of UTI [13]. Five of the 12 accompanying wives had a UTI 2–8 weeks before their husbands’ illnesses. Among 7 male consorts of women with recurring UTI [14], the first-voided urine specimen of 4 grew bacteria with the same serotype as their sex partner.

Families share bowel flora more often than do unrelated persons, but the proportion of shared isolates is small [15]. Among 60 electrophoretic types observed in 655 rectal \( E. \) coli isolates from 5 families, \( E. \) coli of the same electrophoretic type was shared more frequently between members of the same family (11%) than between members of unrelated families in the same city (4.9%) [15]. \( E. \) coli sharing electrophoretic type may differ by PFGE or virulence profile, so the proportion sharing identical isolates by the criteria used in our study might be even smaller.

Uropathogens found in women with UTI can also be present in asymptomatic male sex partners. As our study was cross-sectional, we could not determine the direction or mode of transmission or whether colonized men are at risk of UTI. Although no known bacterial virulence factor, virulence profile, or behavioral factor was associated with colonization of both partners, our numbers are too small to make any definitive conclusions. Larger, prospective follow-up studies are needed to determine what bacterial or behavioral factors (or both) are associated with transmission, the frequency of transmission, and the impact of transmission on the occurrence of symptomatic infection.

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References

Postoperative *Serratia marcescens* Wound Infections Traced to an Out-of-Hospital Source


From 25 August to 28 September 1994, 7 cardiovascular surgery (CVS) patients at a California hospital acquired postoperative *Serratia marcescens* infections, and 1 died. To identify the outbreak source, a cohort study was done of all 55 adults who underwent CVS at the hospital during the outbreak. Specimens from the hospital environment and from hands of selected staff were cultured. *S. marcescens* isolates were compared using restriction-endonuclease analysis and pulsed-field gel electrophoresis. Several risk factors for *S. marcescens* infection were identified, but hospital and hand cultures were negative. In October, a patient exposed to scrub nurse A (who wore artificial fingernails) and to another nurse—but not to other identified risk factors—became infected with the outbreak strain. Subsequent cultures from nurse A’s home identified the strain in a jar of exfoliant cream. Removal of the cream ended the outbreak. *S. marcescens* does not normally colonize human skin, but artificial nails may have facilitated transmission via nurse A’s hands.

*Serratia marcescens* are opportunistic gram-negative bacilli that most commonly cause sporadic urinary tract infections and pneumonias, although outbreaks frequently occur, particularly in intensive care units (ICUs) [1, 2]. The overall national surgical site infection rate for patients undergoing cardiac surgery in the United States during 1990–1992 was 1.1 infections per 100 surgeries [3]. *S. marcescens* was a causative agent in only 1% of surgical site infections [4].

From 25 August to 28 September 1994, 6 of 31 cardiovascular surgery patients and 1 of 29 vascular surgery patients at a community hospital in northern California developed postoperative *S. marcescens* infections. Five patients developed only surgical site infections. Two patients developed bacteremia, 1 of whom died. These were the first *S. marcescens* infections among cardiac or vascular surgery patients at the hospital identified in ≥1 year. The hospital suspended all elective cardiac surgery and requested assistance in its investigation to determine and control the source of the outbreak.

Methods

Cohort Study

We reviewed the medical records of all adults undergoing cardiovascular or vascular surgery (CVS) at the hospital between 16 August (the date of operation for the first patient who developed postoperative *S. marcescens* infection) and 28 September 1994. A case-patient was a cohort member who developed postoperative *S. marcescens* infection, identified by review of microbiology and medical records.

For each patient in the cohort, we examined baseline patient characteristics, preexisting medical conditions, duration and number of invasive procedures, patient locations, exposures to medical staff and ancillary personnel, exposures to various devices and medications, postoperative bathing practices, and number of discharge diagnoses (a proxy for severity of illness).