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Fibrinogen release and deposition on urinary catheters placed during urologic procedures

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Abstract

Purpose—Catheter-associated urinary tract infections (CAUTI) account for ~40% of all hospital-acquired infections worldwide, with more than one million cases diagnosed annually. Recent data from a CAUTI animal model has shown that inflammation induced by catheterization releases host fibrinogen that accumulates on the catheter. Further, *Enterococcus faecalis* catheter colonization was found to be dependent on EbpA, a fibrinogen binding adhesin. We sought to evaluate this mechanism in a human model.

Materials and methods—Urinary catheters were collected from human subjects hospitalized for surgical or non-surgical urologic procedures. Catheters were subjected to immunofluorescence analyses by incubating them with anti-fibrinogen antibody and then stained for fluorescence. The fluorescence intensity was compared to standard catheters. Catheters were incubated with strains of *Enterococcus faecalis, Staphylococcus aureus,* or *Candida* to assess their binding to fibrinogen-laden catheters.

Results—Fifty catheters were collected after various surgical and urological procedures. *In vivo* dwell time ranged from 1 hour to 59 days. All catheters had fibrinogen deposition and its accumulation was dependent on dwell time but not on surgical procedure or catheter material.

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Catheters were probed *ex vivo* with *E. faecalis*, *S. aureus*, and *Candida albicans,* which bound to catheters only in those regions where fibrinogen was deposited.

Conclusions—Taken together, these data show that urinary catheters act as a binding surface for accumulation of fibrinogen, which is released due to inflammation resulting from a urological procedure or from catheter placement, creating a niche that can be exploited by uropathogens to cause CAUTI.

Keywords

Urinary catheterization; CAUTI; fibrinogen; catheter; inflammation

INTRODUCTION

Urinary tract infections (UTIs) represent a significant burden to the healthcare system, affecting approximately 150 million people worldwide[1, 2], with an estimated per annum cost of $2-3 billion in the United States[2]. Serious sequela can result from UTIs including pyelonephritis and bacteremia, resulting in severe morbidity and mortality[3]. Hospitalization is a risk factor for UTI, with an estimated risk on any day of 1.1-6.5%. Catheterized patients have substantially higher risk than patients without exposure to a catheter[4] and virtually all patients will develop bacteriuria within 30 days of catheter placement[1, 5, 6]. Urinary catheters are the second leading cause of hospital-acquired bacteremia leading to 7-day mortality rates of over 30%[7]. Furthermore, the emergence of multidrug resistant microorganisms such as vancomycin-resistant enterococci (VRE), methicillin resistant *Staphylococcus aureus* (MRSA) and fluoroquinolone resistance in *Escherichia coli* is of critical concern[2, 8, 9]. The overuse of antibiotics during UTI can lead to fungal infection and is a universal risk for acquiring *Candida* UTI[10]. Therefore, understanding the interplay between uropathogen virulence factors and the patient response will pave the way for the development of alternative treatments to reduce antibiotic use.

In a murine model of catheter-associated UTI (CAUTI)[11], fibrinogen deposition on catheters is the key pathogenic event leading to colonization by *Enterococcus faecalis*[12]. In this model, circulating fibrinogen increases in response to tissue damage from catheter placement, enters the bladder and is deposited on catheters in a time-dependent manner. *E. faecalis* adheres to the deposited fibrinogen using a hair-like heteropolymeric surface protein, the endocarditis- and biofilm-associated pilus (Ebp)[12-14]. The EbpA subunit located at the tip of the fiber binds fibrinogen to facilitate catheter-associated biofilm formation[12, 15] and vaccination with EbpA attenuates *E. faecalis* colonization and biofilm formation during catheterization[12]. The goal of this study was to translate these observations from the murine model to human disease by: 1) investigating the deposition of fibrinogen on human urinary catheters and; 2) using an *ex-vivo* assay to assess whether fibrinogen deposits can potentiate colonization by several common uropathogens, including *E. faecalis*, *S. aureus* and *Candida albicans.*

METHODS

Bacterial strains and growth conditions

Strains included *E. faecalis* OG1RF (SJH-1994), its isogenic EbpA mutant that can express Ebp, but cannot bind to fibrinogen (SJH-2001)[12], *S. aureus* (SJH-1369) and *Candida albicans* (SJH-3140). Liquid cultures used Brain Heart Infusion broth (BHI) (BD Company) inoculated from a single colony grown on Brain Heart Infusion plates and were grown shaking at 200rpm (*S. aureus, Candida albicans*) or statically (*E. faecalis*) at 37°C for 18 h.

Antibodies

Primary antibodies: Goat anti-human fibrinogen (cat#F8512, Sigma-Aldrich); rabbit anti-Streptococcus group D antigen (anti-*Enterococcus faecalis*) (Lee Laboratories)[12]; rabbit anti-Protein A antigen (anti-*Staphylococcus aureus*) (cat#P3775; Sigma-Aldrich), rabbit anti-*Candida albicans* (cat#ab53891; Abcam). Secondary antibodies: IRDye 800CW donkey anti-goat (cat#926-32213); and IRDye 680LT goat anti-rabbit (cat#926-68021) from LI-COR Biosciences.

Collection of urinary catheters

Urinary catheters consisting of 100% silicone, silicone elastomer or latex were placed for surgical or non-surgical urologic procedures in consented patients (Table 1). The gender, procedure type, catheter material, dwell time, prior urine culture, and antibiotics prescribed were recorded for each patient. This study was approved by the Washington University School of Medicine Internal Review Board (approval #201410058).

Analysis of fibrinogen deposition

After collection, catheters were cut into 10 cm segments, fixed with formalin for 1 hour and washed 3 times with PBS. For analysis, the first 10 cm of the catheter's tip was blocked by overnight incubation at 4°C with 1.5% BSA with 0.1 % sodium azide in PBS, then washed three times for 5 min each with PBS-T (PBS containing 0.05% Tween-20). A 15 ml solution containing goat anti-human fibrinogen diluted 1:500 in PBS-T was added and incubated at room temperature for 2 hrs. Catheters were then washed 3 times for 5 min each with PBS-T, and incubated with donkey anti-goat IRDye 800CW (diluted 1:10,000) for 45 min at room temperature. Following an additional 3 washes with PBS-T, catheters were examined for infrared signal using the Odyssey Imaging System (LI-COR Biosciences). Auto-fluorescence was assessed in non-implanted catheters and catheters not incubated with the primary antibody, which was minimal.

Quantification of fibrinogen

The concentration of fibrinogen deposited on clinical catheters was determined by comparison of the infrared signal following staining to a standard curve prepared by incubation of fibrinogen (0.0001 mg/ml-10 mg/ml) with non-implanted catheters that were then processed as described above. Separate curves were generated for each type of catheter material. A linear relationship model was used to compare the amount of fibrinogen

deposition to variables (dwell times/surgical procedure/bacterial positive urine cultures) that was tested for significance by Pearson's correlation using GraphPad Prism software.

Binding to catheters *ex-vivo*

Catheters from patients with negative urine cultures were treated as described above, except that antibody was replaced with uropathogens as follows: Overnight cultures were washed and resuspended in PBS and cell densities normalized to an OD_{600} of 0.5 (~2 $\times 10^7$ CFU for *E. faecalis* and *S. aureus* strains; ~2×10^4 for *C. albicans*) and 15 ml of the suspension incubated with the patient catheter at 37°C with shaking conditions (100 rpm). Uninfected control catheters were incubated with PBS alone. Following 1 hr, catheters were then washed 3 times with PBS to remove unbound microorganisms, were fixed with formalin for 20 min at room temperature, then washed 3 times with PBS and blocked with 1.5 % BSA and 0.1 % sodium azide in PBS at 4°C overnight. Catheters were then washed 3 times with PBS-T and stained with rabbit antibodies against each pathogen and with goat anti-human fibrinogen (all diluted 1:500), were washed an additional 3 times with PBS-T and incubated with secondary antibodies and scanned for infrared signal as described above. Images were analyzed using Odyssey Infrared Imaging software (version 3.0.16) to measure relative infrared fluorescence at 800 nm and 700 nm, corresponding to fibrinogen and microorganism, respectively. Auto-fluorescence, as determined from non-implanted catheters and catheters incubated only with the secondary antibody, was minimal (typically <0.05% of infected catheters).

RESULTS

Fibrinogen is deposited on human urinary catheters and deposition is dependent on dwell time

Fifty adult patients undergoing urinary catheterization as a standard of care were consented for this study in the Division of Urology at Washington University School of Medicine. Patient gender, the procedure requiring catheterization, catheter type and material, catheter dwell time, urine culture results prior to the procedure (if any), and any antibiotics prescribed are detailed in Table 1. Catheters were collected from both male and female patients undergoing a range of surgical and non-surgical urological procedures and had dwell times from as little as 1 hr to 59 days (Table 1). Silicone catheters were most commonly collected in the study (Fig. 1). We found that regardless of gender, procedure, or catheter material, all catheters stained positive for fibrinogen (Fig. 1). The concentration of fibrinogen deposited on the catheters was quantified (mg/catheter) by using a standard curve for each catheter material (Table 1). Correlation analysis showed that the concentration of fibrinogen was dependent on the dwell time ($r=0.6312$, $p=<0.0001$; Fig. 2A); however, there was not a positive correlation between concentration of fibrinogen deposition and surgical procedure ($r=-0.1181$, $p=<0.4139$) (surgical procedures are grouped by color, Fig. 2A, B). Furthermore, positive preoperative urine cultures do not have a significant correlation with fibrinogen deposition ($r=0.1449$, $p=<0.3153$) (square symbols, Fig. 2A and B). Based on these data, dwell time is the main determinant influencing the concentration of fibrinogen deposition.

Deposited fibrinogen on catheter facilitates colonization by uropathogens

We then investigated whether uropathogens could attach to the fibrinogen deposited on human urinary catheters. For this experiment, we analyzed two strains: i) the *E. faecalis* strain (OG1RF) that expresses the fibrinogen binding EbpA protein and; ii) an isogenic mutant derived from this strain that contains a mutated EbpA protein rendering it unable to bind to fibrinogen (AWAGA)[5, 16]. As an additional comparison, the urinary catheters were mock infected with PBS alone. To monitor auto-fluorescence, control catheters were incubated only with secondary antibodies and no bacteria. The catheters that were used were from patients identified to have negative urine cultures prior to their procedures. Immunofluorescence staining revealed that these catheters had deposited fibrinogen (top, Fig.3A) and that *E. faecalis* OG1RF, but not the fibrinogen-binding mutant (AWAGA), was able to bind to catheters (middle, Fig. 3A). Furthermore, the areas of bacterial adherence co-localized with the pattern of fibrinogen deposition (bottom, Fig. 3A).

Two other prominent uropathogens were similarly tested: i) a UTI MSRA clinical isolate of the gram-positive bacterium *Staphylococcus aureus* and; ii) a kidney stone clinical isolate of the fungal pathogen *Candida albicans*. Both of these species have been reported to encode fibrinogen-binding proteins. Similar to *E. faecalis*, both MSRA (Fig. 2B) and *C. albicans* (Fig. 3C) bound to catheters in patterns that co-localized with deposited fibrinogen. These data suggest that fibrinogen deposition on the catheter creates an ideal environment for microorganisms that are able to exploit fibrinogen during attachment and colonization.

DISCUSSION

In this study, we have shown that fibrinogen is released and deposited on human urinary catheters, where it can facilitate the colonization of uropathogens. These data corroborate results from the murine model of CAUTI to provide important insight into host-pathogen interactions in the pathogenesis of CAUTI.

This study also extends prior reports that human urinary catheterization causes histological and immunological changes in the bladder due to mechanical stress, resulting in a robust inflammatory response, exfoliation, edema of the lamina propria and submucusa, urothelial thinning, and mucosal lesion of the urothelium and kidney[17, 18]. Based on this study, fibrinogen release into the bladder could be caused by the inflammatory response due to the surgical procedure, hematuria, or the placement of the catheter itself. Here we show the presence of a urinary catheter provides a surface for fibrinogen deposition, creating a reservoir that can be exploited by uropathogens to colonize and persist in the bladder[19]. Furthermore, the finding that fibrinogen deposition increases with extended time of catheterization may provide a mechanistic explanation for the lower infection levels that result from clean intermittent catheterization as compared to indwelling or re-use catheterization in patients with urinary retention[20].

Similar to what was shown in the murine model, in this human study, we demonstrated that *E. faecalis* strains exploit the fibrinogen deposited on human catheters in a mechanism that involves recognition of fibrinogen by the EbpA tip adhesin of the Ebp pilus[12]. The N-terminus of EbpA contains conserved domains resembling blood clotting factors and a vWA

domain containing a metal-ion dependent adhesion site (MIDAS)- implicated in fibrinogen binding[12]. Since vaccination with EbpA's fibrinogen-binding domain provides protection against *E. faecalis* in the murine CAUTI model, the data from this study suggests that a similar vaccine may prove efficacious in human catheter-associated infections. This would be particularly valuable to prevent CAUTI by VRE and may help prevent sequela in the healthcare setting.

The observation that fibrinogen promoted colonization by other uropathogens, suggests that a similar strategy may also prove efficacious. For *S. aureus*, at least 20 adhesins that interact with fibrinogen have been identified to date[21]. Several of these have been shown to promote the adherence of *S. aureus* to catheters coated with fibrinogen *in vitro* and to bind to fibrinogen *in vivo*[21]. Among the fungi, *Candida* species are amongst the most common uropathogens[22]. Interesting, like *E. faecalis*, *Candida albicans* adheres only poorly to bladder mucosa, but the risk of a urinary tract infection increases in the presence of an indwelling urinary catheter[10, 23]. It has been shown that *Candida albicans* encodes a fibrinogen-binding protein, Mp58, which is expressed during candidiasis[24, 25]. Thus, the fact that fibrinogen-binding proteins are widely distributed among common uropathogens suggests that fibrinogen-binding may be a common theme in CAUTI pathogenesis. Fibrinogen-binding has not been fully explored in *E. coli*, the most common CAUTI pathogen. However, the genomes of *E. coli* UTI strains typically encode as many as 16 operons encoding chaperone-usher (CUP) pili, each of which likely recognizes a distinct host receptor with stereo-chemical specificity[26, 27]. Thus, determining whether fibrinogen, or some other inflammatory protein that is deposited on catheters, is amongst the repertoire of receptors recognized by *E. coli* CUP pili is a priority. Identification and evaluation of fibrinogen-binding proteins among common CAUTI pathogens will likely identify numerous attractive targets for development of therapeutics.

While the current study has provided an important insight into CAUTI pathogenesis, further work will be required to more generally translate these observations to human disease. For example, while several catheters were obtained from patients 1) with neurogenic bladder or incontinence and 2) undergoing other non-surgical procedures that do not involve urinary manipulation other than catheterization, a limitation of this study was that it primarily included patients undergoing a surgical urological procedure. Thus, the effect of catheterization on bladder inflammation and fibrinogen release should also be evaluated in non-surgical patients or patients undergoing or other surgical procedures. In addition, analysis of catheter-associated fibrinogen deposition in cohorts of CAUTI patients is needed to elucidate the full range of uropathogens associated with fibrinogen binding and their complement of potential fibrinogen-binding proteins. These studies will focus efforts on development of next-generation therapies for treatment of catheter-associated infections.

CONCLUSIONS

Fibrinogen deposits on human urinary catheters in a time-dependent manner, and this event creates a microenvironment for catheter colonization by *E. faecalis, S. aureus* and *C. albicans.* The results of this work will lead future investigations that may focus on interventions by blocking fibrinogen-binding by uropathogens.

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ABBREVIATIONS AND ACRONYMS

UTI	Urinary tract infection
CAUTI	Catheter-associated urinary tract infection
VRE	Vancomycin-resistant enterococci
MRSA	Methicillin-resistant *Staphylococcus aureus*
Ebp	Endocarditis- and biofilm-associated pilus
CUP	chaperon-usher pili
CFU	colony forming unit
RRP	Radical retropubic prostatectomy
PCNL	Percutaneous nephrolithotomy
TURBT	Transurethral resection of bladder tumor
SPT	Suprapubic tube
N/A	not applicable
N/I	urine culture positive but bacteria not identified
Neg	urine culture negative
None	no antibiotics given
U/A	unknown antibiotic treatment
BID	on prescription

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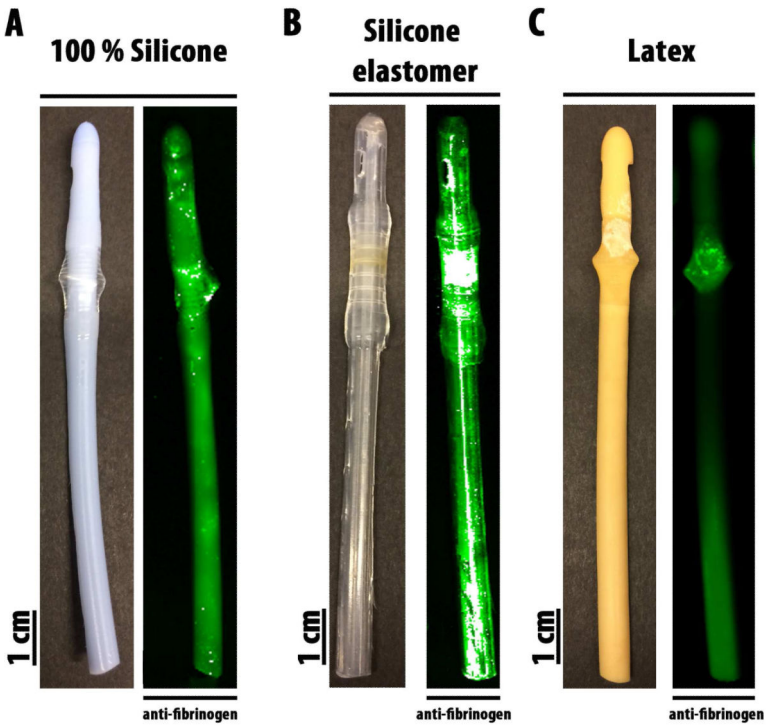

Figure 1. Representative images of the different urinary catheter materials and visualization of their fibrinogen deposition

A) 100 % silicone; B) silicon elastomer; and C) latex. The first 10 cm of the catheter tip was used for fibrinogen deposition analysis. Deposited fibrinogen on the catheter was detected by immunofluorescence using goat anti-human fibrinogen antibody staining.

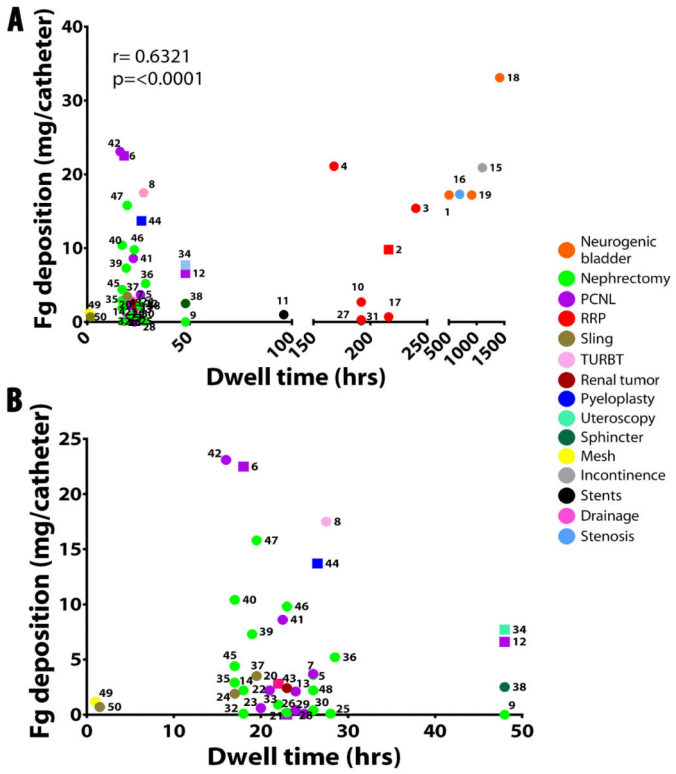

Figure 2. Correlation between the fibrinogen deposition on urinary catheters and their dwell time

(A) All fifty patient urinary catheters placed as a standard of care were recovered and fixed with formalin. The tip section (10 cm) of each catheter was subjected to analysis by immunofluorescence using antibody staining to detect deposited fibrinogen. **(B)** Close up view of the catheters with dwell time up to 50 hrs. Quantification of fibrinogen deposition was calculated by standard curves based on each catheter material. The color represents the surgical procedure that the patient underwent and squares denote those patients that had a preoperative positive urine culture. Pearson' correlation statistical analysis was performed to measure the correlation between fibrinogen deposition and dwell time.

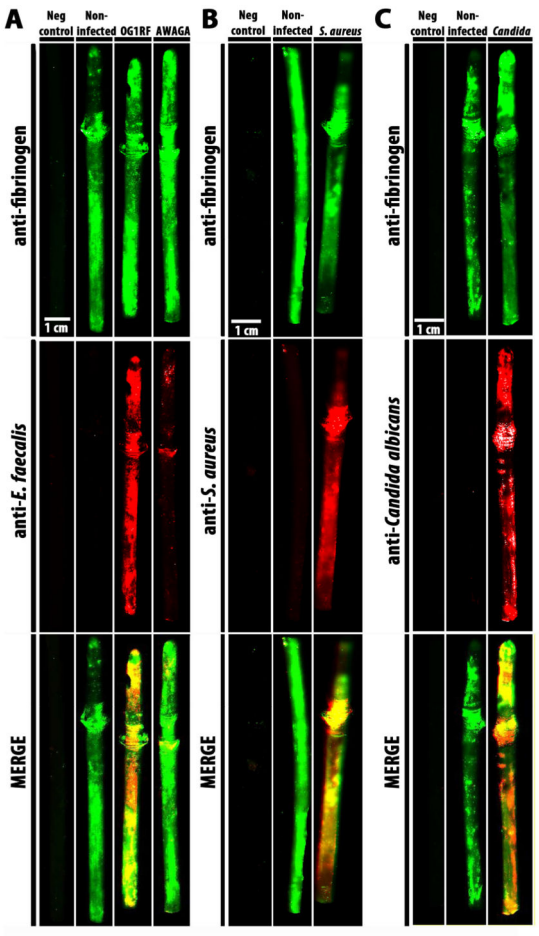

Figure 3. ***Ex-vivo*** **binding of uropathogens encoding fibrinogen-binding proteins to fibrinogen deposited on human urinary catheters**

Urinary catheters from patients with preoperative negative urine culture were used to test the binding of **A)** *E. faecalis*, **B)** *S. aureus,* and **C)** *C. albicans* to fibrinogen deposited onto the catheter. Catheters were incubated with 2×10^7 CFU of the corresponding strain for 1 hour. Detection of the presence and distribution of bacteria and Fg was assessed by immunofluorescent staining. A non-infected catheter was used for each strain for comparison. Catheters incubated only with the secondary antibodies were used as a negative control to assess auto-fluorescence background of the catheter material. Moreover for the *E. faecalis* binding experiment (A), we used the *E. faecalis* wild type (OG1RF) and a known fibrinogen deficient mutant (AWAGA) for comparison.

Table 1

Information on urinary catheters placed as a standard of care.

Pt #	Gender	Procedure	Dwell time	Fg (mg/catheter)	Preop Urine culture	Preop antibiotics	Postop antibiotic	Catheter material
1	F	Neurogenic bladder[#]	21 days	17.2	N/A	None	None	Latex
2	M	RRP	9 days	9.8	Gram +	None	Cipro 500mg	Silicone
3	M	RRP	10 days	15.4	Neg	None	Cipro 500mg	Silicone
4	M	RRP	7 days	21.1	Neg	None	Cipro 500mg	Silicone
5	F	Partial nephrectomy	26 hrs	3.6	Neg	IV Cefazolin 2g	IV Cefazolin 2g	Silicone
6	F	PCNL	18 hrs	22.5	*E. faecalis,* MRSA	IV Cipro 2g IV Vanco 1g	IV Cefepime 2g	Silicone
7	F	PCNL	26 hrs	3.7	Neg	IV Cipro 400mg IV Vanco 1g	IV Cipro 500mg	Silicone
8	M	TURBT	27.5 hrs	17.5	Neg	IV Cipro 400mg	Mytomycin-C	Silicone
9	F	Partial nephrectomy	2 days	0.004	Neg	IV Cefazolin 3g	IV Cefazolin 2g	Silicone
10	M	RRP	8 days	2.7	Neg	None	Cipro 500mg	Silicone
11	F	Stent placement for hydronephrosis	4 days	1.0	N/A	None	None	Silicone
12	F	PCNL	2 days	6.6	*S. aureus*	IV Cefepime 1g IV Vanco 1g	IV Cefepime 2g IV Vanco 1g	Silicone
13	M	PCNL	24 hrs	2.1	Neg	IV Cefazolin 3g	Cipro 500mg	Silicone
14	F	Partial nephrectomy	18 hrs	2.2	Neg	IV Cefazolin 2g	IV Cefazolin 2g	Silicone
15	F	Incontinence[#]	46 days	20.9	N/A	None	None	Latex
16	M	Meatal stenosis and retention [*]	29 days	17.3	N/A	Bactrim DS 500mg BID	None	Latex
17	M	RRP	9 days	0.7	Neg	None	Cipro 500mg	Silicone
18	M	SPT for neurogenic bladder [*]	59 days	33.1	N/A	None	None	Silicone elastomer

Pt #	Gender	Procedure	Dwell time	Fg (mg/catheter)	Preop Urine culture	Preop antibiotics	Postop antibiotic	Catheter material
19	F	SPT for neurogenic bladder *	38 days	17.2	Neg	U/A	U/A	Latex
20	F	Max drainage for r/o sepsis subsequent to pyelonephritis #	22 hrs	2.8	*Actino-baculum*	U/A	U/A	Silicone elastomer
21	M	PCNL	23 hrs	0.004	Coagulase Negative Staphylococci	IV Cipro 400mg IV Vanco 1g	Cipro 500mg BID IV Vanco 1g	Latex
22	F	PCNL	21 hrs	2.2	Neg	IV Cipro 400mg IV Cefazolin 2g	Cipro 500mg BID	Silicone
23	F	PCNL	20 hrs	0.6	Neg	IV Cefazolin 2g	IV Cefazolin 2g	Silicone
24	F	Urethral sling	17 hrs	1.9	Neg	IV Cipro 400mg IV Metronidazole 500mg	Cipro 500mg BID	Silicone elastomer
25	M	Nephrectomy	28 hrs	0.1	Neg	IV Cefazolin 3g	Rifaximin 550mg BID	Silicone
26	M	Radical nephrectomy	23 hrs	0.2	Neg	IV Cefazolin 2g	IV Cefazolin 2g	Silicone
27	M	RRP	8 days	0.2	Neg	None	Cipro 500mg	Silicone
28	M	PCNL	25 hrs	0.1	Neg	IV Cipro 400mg IV Cefazolin 2g	IV Cipro 500mg	Silicone
29	F	PCNL	24 hrs	0.42	Neg	IV Cipro 400mg IV Vanco 100mg	IV Cipro 400mg	Silicone
30	M	Nephrectomy	26 hrs	0.4	Neg	IV clindamycin 900mg	None	Silicone
31	M	RRP	8 days	0.3	Neg	IV Cefazolin 1g	cephalexin 500mg cipro 500mg	Silicone
32	M	Nephrectomy	18 hrs	0.1	Neg	IV Cefazolin 2g	None	Silicone
33	F	Nephrectomy	22 hrs	0.9	Neg	IV Cefazolin 2g	cefazolin 1g	Silicone
34	F	Ureteroscopy	2 days	7.7	*E. coli*	BID BactrimDS 800mg Doxycycine hyclate 100mg	IV cefepime 1g	Latex

Pt #	Gender	Procedure	Dwell time	Fg (mg/catheter)	Preop Urine culture	Preop antibiotics	Postop antibiotic	Catheter material
						IV Cipro 326mg		
35	M	Nephrectomy with stent placement	17 hrs		Neg	IV Cipro 400mg IV Cefazolin 2g	IV Cefazolin 10g	Silicone
36	F	Nephrectomy	28.5 hrs		Neg	IV Cefazolin 2g	IV Cefazolin 1g	Silicone
37	M	Male sling	19.5 hrs	3.5	N/I	IV Vanco 1g	IV Vanco 5 g	Silicone
38	M	Artificial urinary sphincter	23 hrs	2.5	Neg	IV piperacillin/ taxoactam 3.375g/IV vanco 1g	Ampicillin/sulbactam 15g Vanco 5g	Latex
39	M	Radical nephrectomy	19 hrs	7.3	Neg	IV Cefazolin 3g	None	Silicone
40	F	Partial nephrectomy	17 hrs	10.4		IV Cefazolin 2g	None	Silicone
41	F	PCNL	22.5 hrs	8.6	Neg	IV Cefazolin 2g	None	Silicone
42	M	PCNL	16 hrs	23.1	N/A	IV Cefapime 2g IV Vanco 5g	None	Silicone
43	M	Percutaneous resection of right renal pelvic tumor	23 hrs	2.4	Neg	BID cipro 500mg IV gentamycin 300mg IV Cefazolin 2g	None	Silicone
44	M	Right pyeloplasty	26.5 hrs	13.7	*Gardnerella vaginalis*	IV Cefazolin 2g IV Metronidazole 500mg	IV Cefazolin 2g	Silicone
45	M	Partial nephrectomy	17 hrs	4.4	N/I	IV Cefazolin 2g	IV Cefazolin 2g	Silicone
46	M	Partial nephrectomy	23 hrs	9.8	N/I	IV Cefazolin 2g	IV Cefazolin 2g	Silicone
47	M	Partial nephrectomy	19.5 hrs	15.8	Neg	IV Cefazolin 2g	None	Silicone
48	M	Partial nephrectomy	26 hrs	2.2	Neg	IV Cefazolin 2g	None	Silicone
49	F	Excision of eroded mesh	1 hr	1.2	Neg	400mg IV Metronidazole 500mg	None	Silicone elastomer

Pt #	Gender	Procedure	Dwell time	Fg (mg/catheter)	Preop Urine culture	Preop antibiotics	Postop antibiotic	Catheter material
50	F	Mid urethral sling placement	1.5 hrs	0.7	Neg	IV Cefazolin 2g IV Metronidazole 500mg	None	Silicone elastomer

RRP: Radical retropubic prostatectomy

PCNL: Percutaneous nephrolithotomy

TURBT: Transurethral resection of bladder tumor

SPT: Suprapubic tube

N/A: not applicable.

N/I: urine culture positive but bacteria not identified.

Neg: urine culture negative.

None: no antibiotics given.

U/A: unknown antibiotic treatment.

BID: on prescription

[#]: Catheter placement

[*]: Non-surgical procedure