An outbreak of *Pseudomonas aeruginosa* because of inadequate disinfection procedures in a urology unit: A pulsed-field gel electrophoresis-based epidemiologic study

Uner Kayabas, MD,[a] Mehmet Bayraktar, PhD,[b] Baris Otlu, PhD,[b] Murat Ugras, MD,[c] Yasemin Ersoy, MD,[a] Yasar Bayindir, MD,[a] and Riza Durmaz, PhD[b]
Malatya, Turkey

Background: *Pseudomonas aeruginosa* is an opportunistic pathogen causing nosocomial infections in many hospitals. We aimed to investigate the source of urinary tract infections by determining clonal relationship of *Pseudomonas aeruginosa* strains with pulsed-field gel electrophoresis (PFGE).
Methods: During a 2-month period, all postoperative infections because of *P aeruginosa* were investigated in the Urology Department. Patient data were collected from medical records. Surveillance samples were obtained from various places in urological operating rooms. PFGE typing was performed for all *P aeruginosa* isolates.
Results: A total of 14 *P aeruginosa* strains (12 from patients and 2 from environmental samples) were isolated. PFGE typing of these 14 strains yielded 2 possibly related clones, which differed from each other by 4 major bands. Ten of the patient isolates were clonally identical with the strains of 2 forceps.
Conclusion: Typing results confirmed that inadequately disinfected surgical devices can be the source of outbreak. After institution of infection control measures and education, no further clusters of *P aeruginosa* infection were detected in the Urology Department. (Am J Infect Control 2008;36:33-8.)

Pseudomonas aeruginosa is an important gram-negative nosocomial pathogen that exists in humid environment. It can cause a broad spectrum of infections involving the respiratory, gastrointestinal, and urinary tracts as well as wound infections, sepsis, and others.[1] *P aeruginosa* contributes to high morbidity and mortality among the patients in intensive care units, oncology departments, burn units, and surgery wards.[2] Tap water,[1] medical equipment,[3-5] hospital water systems,[6] inadequate disinfection procedures,[3] hospital personnel,[7] and other patients[8,9] are possible sources of *P aeruginosa* infection in hospitals.

From the Department of Infectious Diseases and Clinical Microbiology,[a] Department of Microbiology and Clinical Microbiology,[b] and Department of Urology,[c] Inonu University Medical Faculty, Malatya, Turkey.

Address correspondence to Uner Kayabas, MD, Inonu University, Medical Faculty, Turgut Ozal Medical Center, Department of Infectious Diseases and Clinical Microbiology, 44280, Malatya, Turkiye. E-mail: ukayabas@inonu.edu.tr.

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P aeruginosa usually affects the urinary tract through ascending infection and adheres strongly to bladder uroepithelium.[10] Nosocomial urinary tract infection (UTI) usually results from surgical intervention or instrumentation of the urinary tract involving the prostate gland and urinary bladder. If a cluster of cases is detected, a common source probably associated with urologic instrumentation should be suspected.[2,3,11]

Molecular typing is an important tool to follow transmission routes of microbial pathogens that can be used in clinical settings to discriminate ongoing epidemics of an infectious agent from incidentally increased rates.[12] Even though there are some polymerase chain reaction (PCR)-based typing methods such as arbitrary-primed PCR and amplified fragment-length polymorphism, which are very quick, pulsed-field gel electrophoresis (PFGE) is accepted as the ''gold standard.''[12-14] PFGE has been widely used for clonal analysis of *P aeruginosa* isolates either in a specific setting or population-based research.[8,11-14]

Recently, we noticed that surgery-associated *P aeruginosa* infections increased in the urology ward. Therefore, this study was conducted to trace the source of infections, investigate clonal relationship of strains, and reveal effectiveness of PFGE on taking effective control measures to terminate the outbreak.

METHODS

Setting

This study was carried out in the ward and operating rooms of the Urology Department in a tertiary teaching hospital with 800 beds, 10 intensive care units, and 16 operating rooms. This hospital serves as a referral center for a population of approximately 1,400,000 people. The Urology Department has a ward of 30 beds located on the eleventh floor and 2 central operating rooms located on the subbasement. The urologic team performs approximately 25 elective operations in a week. More than 80% of these operations require endoscopic instrumentation.

Epidemiologic surveillance

Data collection. From March 1 to April 18, 2005, *P aeruginosa* strains were isolated in urine samples of 8 postoperative urology patients, and this situation was described as an outbreak by the Hospital Infection Control Committee. A subcommittee started a surveillance program in the Urology Department on April 18, 2005. Data including demographics, underlying disease, date and type of surgery, surgical procedures, operating room, surgeon and assistants, peri- and postoperative antibiotics, clinical evaluation, type of infection, and outcome were collected from medical records of patients with positive urine cultures for *P aeruginosa*. From April 18 to April 27, 4 additional *P aeruginosa* strains were isolated. Definition of nosocomial infection was made according to the Centers for Disease Control and Prevention definitions.[15]

Environmental sampling and antibiotic susceptibility profiles. By previously informing the urology staff, a total of 22 surveillance samples were obtained from various places of the 2 operating rooms: 14 from equipment including tap outlet of cystoscope and ureterorenoscope, resectoscope loops, inner surface of the pneumatic lithotripter, and forceps and the remaining 8 from the surface of the operation table, vaseline-impregnated gauze, and disinfectant solutions. Samples were taken from both outer surfaces by using moistened cotton swabs and inner surfaces or working channels by flushing with 20 mL of sterile saline. Ten milliliters of each saline wash sample was inoculated into Bactec blood culture bottles (Bactec 9120 System; Becton Dickinson, Sparks, MD) and incubated at 37°C until culture positivity or for 7 days. The moistened swab samples were inoculated directly onto blood agar and eosin-methylene-blue agar and incubated at 37°C for 24 to 48 hours. Two milliliters of disinfectant solution was inoculated directly into Bactec blood culture bottles.

Cultures showing bacteriologic growth were identified on the basis of different conventional biochemical tests. Gram-negative, oxidase-positive, and nonfermentative rods were considered as *Pseudomonas* species. Antimicrobial susceptibility tests for environmental and patients' isolates were performed on Muller Hinton agar using Kirby Bauer methods. The following antimicrobial disks (Oxoid) were used: amikacin (30 μg), gentamicin (10 μg), netilmicin (30 μg), tobramycin (10 μg), mezlocillin (75 μg), aztreonam (30 μg), ceftriaxone (30 μg), ceftazidime (30 μg), cefepime (30 μg), meropenem (10 μg), ciprofloxacin (5 μg), trimethoprim-sulfamethoxazole (1.25/23.75 μg), carbenicillin (100 μg), imipenem (10 μg), and piperacillin (100 μg). *P aeruginosa* ATCC 27853 was used as an internal control. The Clinical and Laboratory Standards Institute criteria were used to determine susceptibility to antimicrobial agents.[16] Identification and antimicrobial susceptibility of the strains were confirmed by an automated system (Phoenix; Diagnostic Instrument Systems; Becton Dickinson).

PFGE

PFGE typing was performed by the method optimized previously by Yetkin et al.[13] Bacterial isolates were grown on nutrient agar overnight, at 37°C. The cells were suspended in 1 mL sodium-EDTA buffer (75 mmol/L NaCl, 25 mmol/L EDTA [pH 8.6]), and the optical density was adjusted to 0.7 ($\lambda = 590$) in spectrophotometer. The cells were embedded into low melting point agarose. After digestion of the cells and washing of the plugs, genomic DNA in the agarose plugs was restricted by 20 U *Xba*I (MBI Fermantas, Hanover, MD) for 6 hours at 37°C in water bath. The separation of DNA fragments was performed in 1.2% pulsed-field certified agarose gel (Bio-Rad Laboratories, Nazareth, Belgium) run in 0.5X Tris-borate-EDTA buffer (44.5 mmol/L Tris, 44.5 mmol/L boric acid, 1 mmol/L EDTA [pH:8.6]) by using a CHEF-DR II system (Bio-Rad Laboratories). The electrophoresis conditions were 11°C at 6 V/cm^2 for 30 hours. The initial and final switch times were 5 seconds and 25 seconds, respectively. The gel was stained with ethidium bromide (5 μg/mL) and photographed under ultraviolet light. According to the interpretative criteria of Tenover et al,[17] isolates were classified as indistinguishable (cluster), closely related, possibly related, or different.

RESULTS

A total of 81 patients underwent endourologic procedures at the urology clinic during the 2-month period, of whom 12 had *P aeruginosa* infections. The patients had a mean age of 51.4 ± 14.8 (range, 23-78) years.

Table 1. Demographic and clinical characteristics of 12 patients who yielded *P aeruginosa*

Pt No	Age/sex	Diagnosis	Operation	Instruments	Indwelling device	Genotype	Infection
1	55/F	Left ureteric stone	Left URS	Cystoscope, ureterorenoscope, forceps	Left DJ stent	A	UTI
2	59/F	Bilateral hydronephrosis	Bilateral RGP and Left URS	Cystoscope, ureterorenoscope,	Bilateral DJ stent	A	UTI
3	40/F	Bilateral kidney stones and left ureteric stone	Right PNL and left URS	Cystoscope, ureterorenoscope, forceps, nephroscope	Left ureter catheter	A1	UTI
4	78/M	BPH and left ureteric stone	TUR-P and left URS	Cystoscope, ureterorenoscope, resectoscope	Left ureter catheter	A1	UTI
5	59/F	Right kidney stone and right ureteric stone	Right PNL and Right URS	Cystoscope, ureterorenoscope, forceps, nephroscope	Right DJ stent	A1	UTI
6	23/M	Left ureteric stone	Left URS	Cystoscope, ureterorenoscope, forceps	Left DJ stent	A1	Sepsis
7	43/M	Left UPJO	Left endopyelotomy	Cystoscope, forceps	Endopyelotomy stent	A1	Sepsis
8	52/M	Bilateral hydronephrosis and right ureteric stone	Bilateral URS	Cystoscope, ureterorenoscope, forceps	Right DJ stent	A1	UTI
9	54/M	BPH	TUR-P	Cystoscope, ureterorenoscope	None	A1	UTI
10	59/M	BPH and left ureteric stone	TUR-P and left URS	Cystoscope, resectoscope, ureterorenoscope, forceps	None	A1	Sepsis
11	63/M	Bilateral ureteric stones	Bilateral diagnostic URS	Cystoscope, resectoscope	Right DJ stent	A1	UTI
12	32/M	Left UVJO	Left URS and left UNC	Cystoscope, ureterorenoscope, open surgical instrumentation	Left DJ stent	A1	UTI

Pt, Patient; *BPH*, benign prostatic hyperplasia; UPJO, ureteropelvic junction obstruction; UVJO, ureterovesical junction obstruction; RGP, retrograde pyelography; *PNL*, percutaneous nephrolithotomy; *TUR-P*, transurethral resection of prostate; *UNC*, ureteroneocystostomy; URS, ureterorenoscopy; UTI, urinary tract infection.

Relevant demographic and clinical data are shown in Table 1. All the patients with positive urine cultures for *P aeruginosa* underwent surgery in the same operating room. The most common operation was ureterorenoscopy (Table 1). *P aeruginosa* growth was observed in 2 forceps tips of 22 environmental samples.

Antibiotic susceptibility tests of 14 (12 patients, 2 forceps) strains revealed that all strains were resistant to trimethoprim-sulfamethoxazole and susceptible to amikacin, gentamicin, ceftazidime, meropenem, imipenem, and ciprofloxacin. Resistance or intermediate susceptibility to carbenicillin, ceftriaxone, mezlocillin, aztreonam, piperacillin, cefepime, netilmicin, and tobramycin was found in 6 (42.9%), 5 (41.7%), 3 (21.4%), 2 (14.3%), 2 (14.3%), 1 (7.1%), 1 (7.1%), and 1 (7.1%) strains, respectively.

Before definitive diagnosis of the outbreak, the first 3 patients were considered as sporadic UTI, and ceftriaxone was begun empirically. Because *P aeruginosa* isolates of these patients were susceptible to ceftriaxone, the therapy was not changed. After outbreak definition, antipseudomonal antibiotics were prescribed for the remaining patients. The initial 3 and the remaining 9 patients (4 with ciprofloxacin, 3 with ceftazidime, 1 with amikasin, and 1 with meropenem) were treated successfully.

PFGE typing of the 14 strains yielded 2 possibly related clones (A and A1), which differed from each other by 4 major bands. Lines 3 and 4 were clone A, and the others were clone A1 (Fig 1). Clone A included 2 patients' isolates, whereas clone A1 included 12 indistinguishable isolates: 10 from patients and 2 from forceps. Clones A and A1 have a similarity coefficient of more than 78% (Fig 1B).

DISCUSSION

In UTIs, horizontal transmission is also implicated, particularly in nursing wards after surgery.[11] In a 2-month period, an outbreak of *P aeruginosa* occurred in patients who underwent endourologic surgery in the Urology Department of our university hospital. Because we considered that an outbreak resulted from the operating room, environmental sampling was performed. *P aeruginosa* was isolated from 2 forceps used in ureterorenoscopy.

It was noted that *Pseudomonas* species, which commonly cause device-related infections, are often resistant to disinfectants.[21] Therefore, medical devices used in surgery such as forceps and cystoscopes should be thoroughly cleaned and sterilized or at least treated with high-level disinfectant adequately.[18] We observed

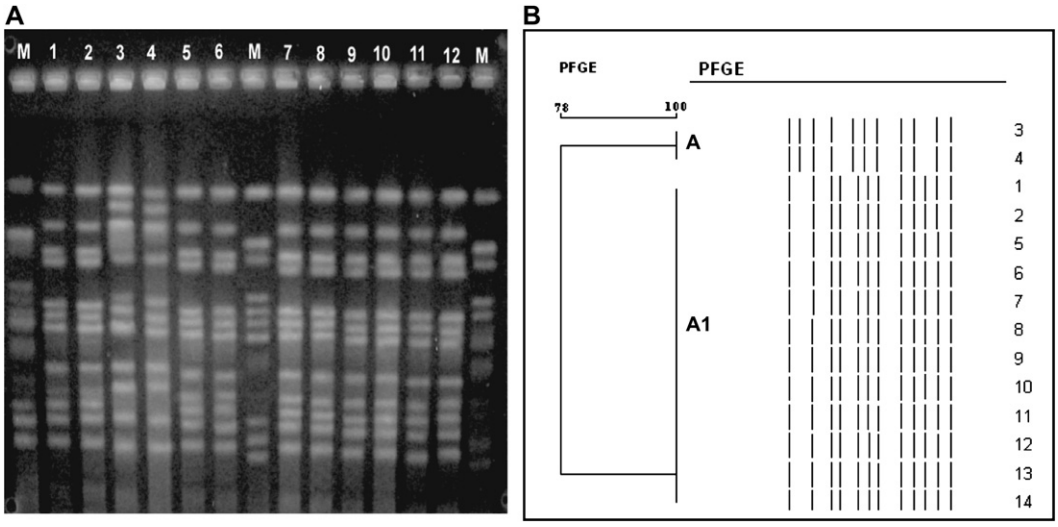

Fig 1. (A) An example of the PFGE profiles of the outbreak strains of *P aeruginosa*. M, *P aeruginosa* ATCC 27853 strain. Lines 1 and 2 included the strains isolated from forceps; lines 3 to 14 were the patients' isolates. (B) Dendrogram of the PFGE results of the 14 *P aeruginosa* strains from patients and forceps. Genotype A included the strains isolated from 2 patients; genotype A1 included the 12 strains isolated from forceps (2 strains) and patients (10 strains).

that this equipment was neither cleaned adequately nor disinfected properly with a solution approved by the US Food and Drug Administration. At the time of outbreak, commercial guanidine derivate disinfectant (Lysetol AF, Schülke&Mayr, Germany),[19] which contains cocospropylene-diamine-guanidine diacetate, phenoxypropanols, and benzalkonium chloride, was used in a concentration of 5%, as proposed by the manufacturer. However, guidelines for usage were not adhered to in 2 points, probably because of workload. First, the solution was used for more than 7 days after preparation. Second, soaking time for equipment was less than 15 minutes. Another source for failure of disinfection was inadequate cleaning of the forceps. Inorganic and organic materials remained on nicked jaw surfaces, which interfered with the efficiency of disinfectant. Several similar outbreaks were reported because of inadequate cleaning and disinfection procedures in clinical practice.[3-5,11]

The forceps were used to grasp stone particles; therefore, they might be contaminated by infected stones (struvites) or other stones carrying bacteria on their surfaces. These forceps are thought to contaminate the working channels of endoscopes, consisting the source of infections in patients who underwent surgery without using forceps. Because these instruments have large working channels that can be cleaned and irrigated easily, microbiologic growth may not be yielded at the time of environmental sampling.

P aeruginosa mainly causes infection in patients who are hospitalized for long periods, undergo medical application, or have underlying diseases.[8,11,20,21] In concordance with these data, all of our patients had endourologic operations by using at least 1 invasive instrument.

Most nosocomial and clinical isolates of *P aeruginosa* are multiresistant.[18,20,21] However, variations have occurred in antibiotic resistance in different countries and even in different hospitals within the same country, probably because of differences in antibiotic usage, sources of infection, and number of tested organisms.[22] Antibiotic resistance has also increased in our hospital[13] and country.[23] In a recent study, it was revealed that throughout the population, clinical isolates of *P aeruginosa* are more virulent as well as more frequently resistant to antibiotics because of acquisition of strains mostly through cross contaminations.[24] High susceptibility of strains in our study supports an environmental source rather than a clinical source.

Molecular techniques for analysis of clinical isolates can be helpful in investigation of the epidemiology of outbreak strains and in confirming their clonality.[5,7,8,11,13,14,25-29] The relevance of exogenous reservoirs and the importance of cross transmission have been convincingly documented during outbreaks.[8] Yetkin et al[13] analyzed clinical and epidemiologic characteristics of the nosocomial *P aeruginosa* infections by testing clonal relationship to emphasize its spread in our hospital previously. In that study, 14 of 80 patients with *P aeruginosa* infections were from the Urology Department, but only 2 isolates were found to be clonally related. On the other hand, PFGE typing results of our

study confirmed that an outbreak resulted from forceps. Because no environmental samples other than from forceps revealed any growth and, furthermore, because the *P aeruginosa* strains of 10 patients were ''indistinguishable'' and strains from 2 other patients were possibly related with the strains of 2 forceps, we considered that forceps contamination was the source of this outbreak. Despite previous reports of outbreaks because of *Klebsiella* species,[26] *Acinetobacter baumannii*,[27] *Chryseobacterium meningosepticum*,[28] and *Stenotrophomonas maltophilia*,[29] this study is the first reported *P aeruginosa* outbreak confirmed by molecular epidemiologic study in our hospital.

To overcome the infection, invasive endoscopic operations were suspended in the Urology Department, surfaces of operating room were cleaned with chlorine solution (Presept Disinfectant Tablet, Johnson & Johnson Company, United Kingdom), instruments were cleaned with both hot water and enzymatic detergent (Endozime AW Plus, The Ruhof Corporation), and then subjected to 2% gluteraldehyde (Cidex Activated Dialdehyde Solution; Advanced Sterılızatıon Products) for at least 20 minutes. Disinfection methodology was emphasized to all workers of Urology Department by Infection Control Committee members. The processes of cleaning and disinfection were strictly followed for 2 months. Bacteriologic reassessment was done afterwards, revealing no pathogens. The operations were then allowed, and no further case of infection with this strain was detected.

In conclusion, nosocomial outbreak should be identified immediately, and infection control measures should be carried out adequately. Molecular typing methods can help in understanding the route of contamination and reveal unsuspected problems of either environmental or hospital staff origin. In this way, obscured causes of contamination such as the lack of compliance with infection control procedures may be demonstrated. To prevent emergence of new outbreaks, the policy of disinfection and sterilization should be followed strictly, and sustained education should be obtained.

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