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Microbiological Characteristics of Acute Prostatitis After Transrectal Prostate Biopsy

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Purpose: We aimed to identify microbiological characteristics in patients with acute prostatitis after transrectal prostate biopsy to provide guidance in the review of prevention and treatment protocols.

Materials and Methods: A retrospective analysis of medical records was performed in 1,814 cases who underwent prostate biopsy at Seoul St. Mary's Hospital and St. Vincent's Hospital over a 5 year period from 2006 to 2011. Cases in which acute prostatitis occurred within 7 days after the biopsy were investigated. Before starting treatment with antibiotics, sample collections were done for culture of urine and blood. Culture and drug susceptibility was identified by use of a method established by the Clinical and Laboratory Standards Institute.

Results: A total of 1,814 biopsy procedures were performed in 1,541 patients. For 1,246 patients, the procedure was the first biopsy, whereas for 295 patients it was a repeat biopsy. Twenty-one patients (1.36%) were identified as having acute bacterial prostatitis after the biopsy. Fifteen patients (1.2%) had acute prostatitis after the first biopsy, and 6 patients (2.03%) experienced acute prostatitis after a repeat biopsy. Even though the incidence of acute bacterial prostatitis was higher after repeat biopsy than that after the first biopsy, there was no statistically significant intergroup difference in terms of incidence (χ^2=1.223, p=0.269). When the collected urine and blood samples were cultured, *Escherichia coli* was found in samples from 15 patients (71.4%), *Klebsiella pneumoniae* in 3 patients (14.3%), *Enterobacter intermedius* in 1 patient (4.8%), *E. aerogenes* in 1 patient (4.8%), and *Pseudomonas aeruginosa* in 1 patient (4.8%). A fluoroquinolone-resistant strain was confirmed in 5 cases (23.8%) in total. Three cases of *E. coli* and 1 case of *Klebsiella* had extended-spectrum β-lactamase activity.

Conclusions: Empirical treatment of acute prostatitis should be done with consideration of geographical prevalence and drug resistance. This study will provide meaningful information for the management of acute prostatitis after transrectal prostate biopsy.

Keywords: *Acute diseases; Beta-lactamases; Biopsy; Fluoroquinolone; Prostate*

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INTRODUCTION

Transrectal ultrasound (TRUS)-guided needle biopsy is the standard diagnostic method for diagnosing prostate cancer. The risks and complications of TRUS-guided needle biopsy are widely known; these are usually minor problems such as hematuria or hematospermia, but complications such as urinary tract infection and sepsis often have very serious consequences. Bacterial sepsis is the most serious complication. The postbiopsy incidence of bacteremia is in the range of 16% to 73%, whereas the incidence of bacteriuria is in the range of 35% to 44% [1-3]. In addition, fatal

TABLE 1. Patients' characteristics

Characteristic	Seoul St. Mary's Hospital (935 biopsies)		St. Vincent's Hospital (879 biopsies)		Total (1,814 biopsies)		Overall
	First	Repeat	First	Repeat	First	Repeat	
No. of patients	622	174	624	121	1,246	295	1,541
Mean age (y)	61.2	64.5	65.7	63.3	63.5	63.9	63.7
Median PSA (ng/mL)	7.78	6.91	9.15	8.72	8.47	7.82	8.14
Acute prostatitis (%)	7 (1.13)	3 (1.72)	8 (1.28)	3 (2.48)	15 (1.20)	6 (2.03)	21 (1.36)

PSA, prostate-specific antigen.

septic shock is sometimes induced after prostate biopsy [4,5]. The most commonly identified bacteria in urine culture and blood culture is *Escherichia coli* [1,2]. Administration of prophylactic antibiotics before biopsy has been widely used as a way to prevent serious infection-related complications, and a recent study showed that fluoroquinolone is the most effective treatment [6-8]. However, in some cases, fluoroquinolone-resistant infection has been reported because of the increase of bacterial strains resistant to the broad-spectrum antibiotic and, especially in recent years, the emergence of resistant strains that demonstrate extended-spectrum β-lactamase (ESBL) activity. In the present study, we aimed to identify microbiological characteristics in patients with acute prostatitis incurred after transrectal prostate biopsy to provide guidance in the review of prevention and treatment protocols.

MATERIALS AND METHODS

A retrospective analysis of medical records was performed in 1,814 cases (Seoul St. Mary's Hospital, 935 cases; St. Vincent's hospital, 879 patients) who underwent prostate biopsy over a 5-year period from 2006 to 2011. Indications for prostate biopsy were an elevation of prostate-specific antigen or palpable nodules in the prostate noted during a digital rectal examination. All prostate biopsy procedures were conducted transrectally as 10-core biopsies by use of an automatic biopsy gun with an 18-gauge needle. For prophylactic antibiotics, a single injection of Flomoxef (Flumarin) 500 mg was injected intravenously before the start of biopsy and subsequently 100 mg of Cefcapene (Flomox) was orally administered three times daily for 3 days. Cases in which acute prostatitis occurred within 7 days after the biopsy were investigated. The symptoms of a fever over 38°C, leukocytes in urine sediment, and tenderness of the prostate during digital rectal examination were defined as acute prostatitis. Before treatment with antibiotics was started, sample collections were done for culture of urine and blood, and the minimal inhibitory concentration (MIC) was measured by the broth microdilution method with use of the Clinical and Laboratory Standards Institute Criteria. Drug susceptibility was identified with the breakpoint MIC by use of a method established by the Clinical and Laboratory Standards Institute. The chi-square test was used for statistical processing, and p-values of <0.05 were considered statistically significant. The Institutional Review Board of The Catholic University of Korea College of Medicine, approved the study protocol (SC12RISI0011, VC12RIMI0005).

RESULTS

From January 2006 to December 2011, 1,814 biopsy procedures were performed in 1,541 patients. For 1,246 patients, the procedure was the first biopsy, whereas for 295 patients it was a repeat biopsy. Patient demographics and bacteriologic findings are described in Table 1 and Table 2, respectively.

Twenty-one patients (1.36%) were identified as not having any other prebiopsy urinary tract infection or symptoms of acute prostatitis but as having acute bacterial prostatitis after the biopsy. Of these patients, 15 patients (1.2%) developed acute prostatitis after the first biopsy and 6 patients (2.03%) developed it after a repeat biopsy. Among the patients who were diagnosed with acute bacterial prostatitis after a repeat biopsy, none had experienced the same problem at a previous biopsy. Even though the incidence of acute bacterial prostatitis was higher after a repeat biopsy than after the first biopsy, there was no statistically significant intergroup difference in incidence (χ^2=1.223, p=0.269).

The median age of the patients within the category was 63.7 years (range, 52 to 77 years). Patients had shown symptoms from 2 days after biopsy on average. All patients had a high fever ($\geq$38°C), and 15 patients (71.4%) showed leukocytosis (white blood cell $>$ 10,000 cells/mL). All patients were admitted to the hospital and administered intravenous antibiotics. According to the culture identification results, ceftriaxone was used in 8 patients, fluoroquinolone in 4 patients, amikacin in 5 patients, and piperacillin/tazobactam in 4 patients. No cases resulted in septic shock or death.

All cases showed positive results for urine culture, whereas 42.8% (9/21) of patients showed positive results for blood culture. When the collected urine and blood samples were cultured, *E. coli* was found in samples from 15 patients (71.4%), *Klebsiella pneumoniae* in 3 patients (14.3%), *Enterobacter intermedius* in 1 patient (4.8%),

TABLE 2. Microbiological details of patients

Biopsy no.	Culture		Bacteria	ESBL	Susceptibility													
	Urine	Blood			AMK	AUG	AMPC	AZT	CFPM	CTX	CFXT	CEZ	GM	TOBV	IPM	LVFX	TAZC	TMP/SMX
First	+	+	*Escherichia coli*	-	S	S	S	S	S	S	S	S	S	S	S	S	S	S
First	+	-	*Escherichia coli*	-	S	S	S	S	S	S	S	S	S	-	S	S	S	S
First	+	+	*Klebsiella pneumoniae*	-	S	-	R	S	S	S	S	S	S	-	S	S	S	S
First	+	-	*Enterobacter intermedius*	-	S	-	S	S	S	S	S	R	S	S	S	S	S	S
First	+	-	*Escherichia coli*	-	S	-	R	S	S	S	S	I	S	S	S	S	S	S
First	+	+	*Escherichia coli*	-	S	-	R	S	S	S	S	S	S	S	S	R	S	R
First	+	+	*Escherichia coli*	+	S	S	R	R	R	R	S	R	S	S	S	S	S	R
First	+	-	*Enterobacter aerogenes*	-	S	R	R	S	S	S	R	R	R	S	S	S	S	S
First	+	+	*Escherichia coli*	-	S	-	R	S	S	S	S	I	S	S	S	S	S	S
First	+	-	*Escherichia coli*	-	S	-	R	S	S	S	S	S	S	S	S	S	S	S
First	+	-	*Klebsiella pneumoniae*	+	S	S	R	R	R	R	S	R	R	S	S	R	S	R
First	+	+	*Escherichia coli*	-	S	-	R	S	S	S	S	S	R	R	S	R	S	R
First	+	-	*Escherichia coli*	-	S	S	R	S	S	S	S	R	S	S	S	S	S	S
First	+	-	*Pseudomonas aeruginosa*	-	S	-	-	S	S	I	-	-	S	S	S	S	S	S
First	+	+	*Escherichia coli*	-	S	I	R	S	S	S	S	I	S	S	S	S	S	R
Second	+	-	*Escherichia coli*	+	S	S	R	R	R	R	S	R	S	S	S	R	S	S
Second	+	-	*Escherichia coli*	-	S	S	R	S	S	S	S	I	S	S	S	S	S	R
Second	+	+	*Klebsiella pneumoniae*	-	S	S	R	S	S	S	S	S	S	S	S	S	S	S
Second	+	-	*Escherichia coli*	+	S	S	R	R	R	R	S	R	R	S	S	R	S	R
Second	+	+	*Escherichia coli*	-	S	I	R	S	S	S	S	S	S	S	S	S	S	S
Third	+	-	*Escherichia coli*	-	S	S	S	S	S	S	S	S	S	S	S	S	S	S

ESBL, extended-spectrum β-lactamase; AMK, amikacin; AUG, amoxicillin-clavulanic acid; AMPC, ampicillin; AZT, aztreonam; CFPM, cefepime; CTX, cefotaxime; CFXT, cefoxitin; CEZ, cefazolin; GM, gentamycin; TOBV, tobramycin; IPM, imipenem; LVFX, levofloxacin; TAZC, piperacillin/tazobactam; TMP/SMX, trimethoprim/sulfamethoxazole; S, sensitive; R, resistant; I, intermediate.

TABLE 3. Susceptibility of isolated *Escherichia coli* (n=15)

Antibiotic	Susceptibility (%)
Amikacin	100.0
Amoxicillin-clavulanic acid	80.0
Ampicillin	20.0
Aztreonam	80.0
Cefotaxime	80.0
Cefoxitin	100.0
Cefazolin	46.7
Gentamycin	86.7
Tobramycin	92.9
Imipenem	100.0
Levofloxacin	73.3
Piperacillin/tazobactam	100.0
Trimethoprim/sulfamethoxazole	60.0

Enterobacter aerogenes in 1 patient (4.8%), and *Pseudomonas aeruginosa* in 1 patient (4.8%). A fluoroquinolone-resistant strain was confirmed from 5 cases (23.8%) in total, and 3 cases of *E. coli* and 1 case of *Klebsiella* were ESBL(+). The antibiotic susceptibility of *E. coli* is described separately in Table 3.

DISCUSSION

Administration of prophylactic antibiotics before transrectal prostate biopsy significantly reduces the likelihood of urinary tract infection. However, despite the use of prophylactic antibiotics, fever of 38 degrees or more occurs in 1% to 5% of cases [6,9-12]. Many drugs have been proposed for prophylactic use, and Taylor and Bingham [8] summarized these to 13 different antibiotics. In general, because fluoroquinolone has higher bioavailability in the prostate, this drug family is the most commonly used in transrectal biopsy for the purpose of prophylactic antibiotics [6,8, 11-13]. However, some studies have reported patients developing fluoroquinolone-resistant infections after prostate biopsy. Tal et al. [14] also reported that fluoroqui-"nolone-resistant *E. coli* was the most critical cause of urinary tract infection incurred after transrectal prostate biopsy, and Otrock et al. [15] reported that 50% of patients were admitted to the hospital and treated for urinary tract infection owing to fluoroquinolone-resistant *E. coli* incurred after transrectal prostate biopsy.

Feliciano et al. [16] reported that in postbiopsy acute prostatitis, the incidence of fluoroquinolone-resistant *E. coli* was 86% and that fluoroquinolone resistance appeared to increase as shown by a survey of over 1,273 patients for 2 years. In other research, all isolated *E. coli* was resistant to levofloxacin, although the relevant cases were small in number [17]. In Korea, the rate of fluoroquinolone-resistant pathogens in postbiopsy acute prostatitis was reported to be 96.3% [18].

Almost all of the above-mentioned studies debated the use of fluoroquinolone as a prophylactic antibiotic in transrectal prostate biopsy. As a necessity, the microbiological cause of postbiopsy acute prostatitis is subject to show a high rate of resistance to fluoroquinolone.

The results of the present study on microbiological characteristics after transrectal prostate biopsy are similar to the results of previously reported studies in terms of the overall infection rate and the variety of isolated pathogens [14-17,19]. Regarding antibiotic resistance, however, there were considerable differences between our results and those of previous studies. In this study, fluoroquinolone resistance was expressed in 23.8% (5/21) of strains, which differs from the previous study of postbiopsy acute prostatitis but is similar to another *E. coli*-related general urinary tract infection study that reported that the fluoroquinolone resistance rate was approximately 20% to 30%. This fact is broadly similar to other Korean data concerning acute prostatitis contingent upon urological manipulation in which a resistance rate of 28.6% of *E. coli* to ciprofloxacin was reported [20]. In the hospitals where this study was conducted, fluoroquinolone was not frequently used as a prophylactic antibiotic after 2006; flomoxef is used at present. This may be one of the reasons the postbiopsy acute prostate did not show a high fluoroquinolone resistance rate, unlike other prostate studies but similar to other general urinary tract infection studies.

The wide use of these medications is considered to be the reason for the increase in fluoroquinolone resistance in the first place. In addition, some studies reported that an increased expression of quinolone-resistant *E. coli* was found in the stool of patients who were treated with fluoroquinolone prophylaxis [21]. Shigehara et al. [17] suggested that the previous use of levofloxacin triggered bacterial selection inside the rectum, which led to the emergence of levofloxacin-resistant *E. coli*. This phenomenon has something in common with the study of Ha et al. [22], who reported that the effectiveness of ciprofloxacin was low in patients with a prior history of urological manipulation.

The use of aminoglycoside or cephalosporin as prophylactic antibiotics before prostate biopsy has been researched in a few studies, but these agents did not show significant superiority over fluoroquinolone [23-26]. Because it is highly likely that fluoroquinolone-resistant *E. coli* may increase further, not only the use of fluoroquinolone as a preprostate biopsy prophylactic antibiotic but also the use of other antibiotics such as high-dose aminoglycoside or cephalosporin should be considered. Our study may serve to provide basic information for various prophylactic antibiotics research.

When the difference between the first biopsy and a repeat biopsy was reviewed, the frequency of acute prostatitis was somewhat higher in the repeat biopsy group (2.03% vs. 1.20%), but the difference was not statistically significant. This outcome is similar to a previous report about postbiopsy complications [10,17,22]. The results of our study may have some distinctions from other studies of first and repeat biopsy complications in terms of the methods used for prophylactic antibiotic treatment.

Other than the aforementioned, many studies are being

conducted on the emergence of ESBL-producing *E. coli*. ESBL is an enzyme that neutralizes broad-spectrum antibiotics such as the third-generation cephalosporins or monobactams. Bacteria including *E. coli* and *Klebsiella pneumonia* that often cause urogenital tract infection are potentially ESBL-producing microorganisms. The existence of ESBL-producing organisms can cause therapeutic failure in infectious diseases. In our study, 19.0% (4/21) of bacteria were ESBL-producing strains. The emergence of ESBL activity is thought to be related to the high frequency of use of broad-spectrum antibiotics [27,28]. It is very important to make an accurate differentiation of ESBL-producing microorganisms. ESBL-producing *E. coli*-induced bacteremia has far higher mortality than non-ESBL-producing *E. coli*-induced bacteremia [29,30]. In our study, it would be difficult to directly identify the prevalence of ESBL activity or its sequelae because the study did not include many cases with postbiopsy acute prostatitis. In the future, more extensive research is needed to determine the effects of ESBL activity.

CONCLUSIONS

In acute prostatitis after transrectal prostate biopsy, it is essential to administer appropriate antibiotics immediately. Recently, however, more cases are caused by fluoroquinolone-resistant microorganisms or ESBL-producing microorganisms. Therefore, any empirical treatment should take into account geographical prevalence and drug resistance. To achieve this, microbiological data should be collected to optimize clinical guidelines. These efforts are essential to reduce the indiscriminate use of antibiotics. This study will provide meaningful information for the management of acute prostatitis after transrectal prostate biopsy.

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