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Alarming Quinolone and Fluoroquinolone Resistance in Uropathogenic *E. Coli* from Acute Prostatitis Cases in Niger: A Call for Urgent Empiric Treatment Re-Evaluation

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Abstract **Original Research Article**

Background: The management of acute prostatitis is increasingly complicated by the growing antibiotic resistance of causative pathogens. This study aimed to determine the resistance profiles of uropathogenic Escherichia coli isolates to quinolones and fluoroquinolones in patients with acute prostatitis in Niger. ***Methods:*** A prospective cross-sectional study was conducted from July 1 to October 15, 2018, at the Lamordé National Hospital in Niamey. Urine samples from patients clinically diagnosed with acute prostatitis underwent cytobacteriological examination and culture. Phenotypic identification of bacterial isolates and antibiotic susceptibility testing were performed using the Kirby-Bauer disk diffusion method, following CA-SFM/EUCAST 2017 guidelines. ***Results:*** Of 223 patients seen in the urology department during the study period, 73 (32.7%) were diagnosed with acute prostatitis. Among these, 65 (89%) had positive urine cultures. E. coli was the predominant pathogen, accounting for 33 (50.77%) of all isolates. Susceptibility testing revealed an extremely high resistance rate of E. coli to nalidixic acid (a quinolone) at 96.9%. Resistance to fluoroquinolones was also alarmingly high, with 60.6% of isolates resistant to ciprofloxacin and 48.5% to ofloxacin. ***Conclusion:*** The high prevalence of quinolone and fluoroquinolone resistance in E. coli causing acute prostatitis necessitates an urgent revision of empirical treatment guidelines in Niger. These findings underscore the critical need for routine antimicrobial surveillance to guide therapeutic choices. Further molecular studies are warranted to characterize the underlying resistance genes and mechanisms.

Keywords: Antibiotic Resistance; Escherichia coli; Quinolones; Fluoroquinolones; Acute Prostatitis; Niger.

1. INTRODUCTION

Urinary tract infections (UTIs) result from the invasion of urinary system tissues by one or more microorganisms, triggering an inflammatory response of varying intensity. These infections are most commonly caused by bacteria originating from the host's intestinal or perineal flora (Epok *et al.,* 1999; Adonis *et al.,* 2003). UTIs are among the most frequent community-acquired infections, with Enterobacteriaceae being the primary causative agents (Wiener, 2016).

Escherichia coli, a Gram-negative bacillus of the Enterobacteriaceae family, is a commensal bacterium in the human gut. While typically harmless and beneficial, it can become pathogenic under certain conditions. Pathogenic *E. coli* strains are associated with a wide range of intestinal and extra-intestinal diseases. Their pathogenic strategy involves mucosal colonization, potential cell invasion, multiplication, evasion of host defenses, and host tissue damage (Marie-Pierre, 2009). When displaced from the intestinal tract, *E. coli* can colonize other sites, such as the urinary tract, where uropathogenic strains can cause infection.

In Europe, *E. coli* is responsible for 50-80% of community-acquired UTIs (Epok *et al.,* 1999). A multicenter study in France on male UTIs found *E. coli* to be predominant in 82% of cases (Paumier *et al.,* 2022). Similarly, studies in North Africa and Côte d'Ivoire have reported high prevalence rates of *E. coli* in UTIs (2010; Koffi *et al.,* 2003).

Antimicrobial resistance is a major global public health crisis. The emergence of community-acquired strains producing extended-spectrum β-lactamases (ESBLs), which are often co-resistant to fluoroquinolones and trimethoprim-sulfamethoxazole, is a growing concern (Wiener, 2016). Infections caused by such multidrug-resistant organisms are associated with an increased risk of therapeutic failure, prolonged hospital stays, and higher healthcare costs (Rodriguez *et al.,* 2006). The ability of *E. coli* to acquire and transfer resistance genes via mobile genetic elements like plasmids and phages exacerbates this problem (Together, 2019).

In developing countries like Niger, where antibiotic use is often poorly regulated, the challenge of resistance is particularly acute. The increasing failure of first-line empirical treatments for infections like acute prostatitis highlights the need for etiological diagnosis and susceptibility testing. However, data on antimicrobial resistance patterns for UTIs in Niger are scarce. This study was therefore conducted to investigate the bacterial etiology of acute prostatitis, with a focus on the resistance profile of *E. coli* to quinolones and fluoroquinolones, to provide crucial data for improving diagnosis and treatment strategies.

2. MATERIALS AND METHODS

2.1. Study Design and Setting

A prospective cross-sectional study was conducted over a four-month period, from July 1 to October 15, 2018. The study was carried out at the urology and biology departments of the Lamordé National Hospital in Niamey, a major referral center for the capital city and surrounding regions of Niger.

2.2. Study Population and Sampling

All patients presenting with clinical symptoms suggestive of acute bacterial prostatitis were eligible for inclusion. Inclusion criteria also required the presence of significant leukocyturia and bacteriuria. A non-probabilistic convenience sampling method was used, resulting in a final sample size of 73 patients who met the inclusion criteria.

2.3. Sample Collection and Processing

Urine was the biological material for this study. Samples were collected after obtaining patient consent and completing a data collection form. For patients with urinary retention, urine was obtained via suprapubic aspiration. For patients with an indwelling catheter, urine was collected aseptically from the catheter port. Samples were collected in sterile dry tubes and transported to the laboratory within 30 minutes for cytobacteriological examination, which was performed within two hours of collection.

2.4. Cytobacteriological Examination of Urine

Macroscopic and Microscopic Examination: Each urine sample was first examined for macroscopic appearance. A fresh, centrifuged urine sediment was then examined microscopically to semi-quantitatively assess for leukocytes, erythrocytes, epithelial cells, crystals, casts, and microorganisms. Samples with a high leukocyte count underwent quantitative cytology.

Cytology: Leukocytes were counted using a Malassez counting chamber. A count of ≥10,000 leukocytes/mm³ (or ≥10/mm³) was considered indicative of a UTI (Bourlet *et al.,* 2015).

Gram Stain: A Gram stain was performed on the urine sediment to differentiate between Gram-positive and Gram-negative bacteria and to observe bacterial morphology.

2.5. Bacterial Culture and Identification

Urine samples with significant leukocyturia and bacteriuria were inoculated onto Cysteine Lactose Electrolyte Deficient (CLED) agar and Bromocresol Purple (BCP) agar. A 10 µL loop was used to inoculate the plates, which were then incubated at 37°C for 18-24 hours. A bacterial count of ≥10³ CFU/mL for *E. coli* was considered significant bacteriuria in the context of clinical symptoms (Bourlet *et al.,* 2015). Bacterial isolates were identified based on colony morphology, Gram stain characteristics, and biochemical tests, including oxidase, catalase, and API (Analytical Profile Index) galleries.

2.6. Antibiotic Susceptibility Testing

Antimicrobial susceptibility was determined using the Kirby-Bauer disk diffusion method on Mueller-Hinton (MH) agar. The selection of antibiotic disks and the interpretation of inhibition zone diameters were performed according to the 2017 guidelines of the Antibiogram Committee of the French Society for Microbiology (CA-SFM/EUCAST).

3. RESULTS

3.1. Patient Demographics and Culture Results

During the study period, 223 patients were consulted in the urology department. Of these, 73 (32.7%) were diagnosed with acute prostatitis. Urine cultures were positive in 65 (89%) of these cases, while 8 (11%) showed no bacterial growth.

3.2. Frequency of Isolated Bacteria

Enterobacteriaceae constituted over 90% of the isolates. *Escherichia coli* was the most frequently isolated pathogen, accounting for 50.77% (n=33) of all positive cultures. Other isolated bacteria included *Klebsiella pneumoniae* (15.38%), *Klebsiella oxytoca* (12.31%), and *Serratia odorifera* (7.69%). The complete distribution of isolated pathogens is shown in Table 1.

Table 1

Isolated Organism	Number of Isolates	Percentage (%)
Enterobacteriaceae		
Escherichia coli	33	50.77
Klebsiella pneumoniae	10	15.38
Klebsiella oxytoca	8	12.31
Serratia odorifera	5	7.69
Enterobacter cloacae	3	4.62
Non-Enterobacteriaceae		
Pseudomonas aeruginosa	4	6.15
Acinetobacter baumannii	1	1.54
Staphylococcus aureus	1	1.54
Total	**65**	**100**

3.3. Antibiotic Susceptibility Profile of Escherichia coli

The susceptibility testing of the 33 *E. coli* isolates revealed extremely high levels of resistance to quinolones and fluoroquinolones. Resistance to nalidixic acid (a first-generation quinolone) was observed in 96.9% of isolates. For the fluoroquinolones, 60.6% of isolates were resistant to ciprofloxacin and 48.5% were resistant to ofloxacin. All isolates remained 100% susceptible to carbapenems (imipenem) and amikacin. The detailed susceptibility profile is presented in Table 2.

Table 2: Antibiotic Susceptibility Profile of *Escherichia coli*

Antibiotic (Disk content)	Susceptible (n)	Intermediate (n)	Resistant (n)
Amoxicillin/Clavulanic acid (AMC, 20/10 µg)	10	7	16
Cefuroxime (CXM, 30 µg)	1	18	14
Cephalothin (CF, 30 µg)	3	15	15
Cefotaxime (CTX, 5 µg)	12	4	17
Ceftriaxone (CRO, 30 µg)	12	9	12
Aztreonam (AT, 30 µg)	33	0	0
Imipenem (IPM, 10 µg)	33	0	0
Amikacin (AN, 30 µg)	33	0	0
Netilmicin (NET, 10 µg)	23	3	7
Nalidixic Acid (NA, 30 µg)	**1**	**0**	**32**
Ciprofloxacin (CIP, 5 µg)	**13**	**0**	**20**
Ofloxacin (OFX, 5 µg)	**17**	**5**	**11**
Trimethoprim/Sulfamethoxazole (SXT, 1.25/23.75 µg)	4	9	20
Fosfomycin (FF, 200 µg)	23	3	7
Nitrofurantoin (FU, 100 µg)	13	6	14

3.4. Quinolone Resistance Phenotypes

Among the *E. coli* isolates, the ciprofloxacin-resistant (CipR) phenotype was the most common, observed in 42.42% of strains. The distribution of phenotypes based on ciprofloxacin susceptibility is shown in Figure 1.

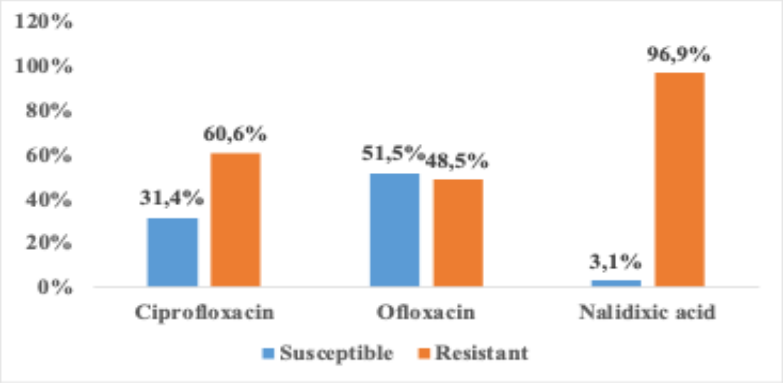

Figure 1: Distribution of Quinolones Resistance Phenotypes in E. coli Isolates (n=33). Cip: Ciprofloxacin; OFX: Ofloxacin: NA: Nalidixic Acid.

4. DISCUSSION

This study provides critical, contemporary data on the etiology and antimicrobial resistance patterns of acute prostatitis in Niamey, Niger. The finding that *E. coli* is the predominant pathogen (50.77%) is consistent with global and regional literature, confirming its primary role in male UTIs (Wiener, 2016; Fabry *et al.,* 2010).

The most striking finding of this study is the alarmingly high level of resistance to quinolones and fluoroquinolones. A resistance rate of 96.9% to nalidixic acid and 60.6% to ciprofloxacin renders these agents empirically useless for treating acute prostatitis in this setting. Fluoroquinolones have long been considered a first-line treatment for prostatitis due to their excellent prostatic tissue penetration. The high resistance rates observed here suggest widespread therapeutic failure is likely occurring when these drugs are used empirically.

These resistance levels are significantly higher than those reported in many European countries but are becoming increasingly common in regions with less regulated antibiotic access. The widespread availability and overuse of these antibiotics in both human and veterinary medicine in many developing nations likely drives the selection and spread of resistant strains. The high resistance to trimethoprim-sulfamethoxazole (60.6% resistant) further limits oral therapeutic options.

In contrast, the isolates showed 100% susceptibility to imipenem and amikacin. While effective, these are injectable agents, typically reserved for severe or complicated infections, and are not suitable for routine outpatient management of uncomplicated prostatitis. The relatively good susceptibility to fosfomycin (7.6% resistance) and nitrofurantoin (42.4% resistance) suggests these may be alternative options, although nitrofurantoin has poor prostatic penetration and is not recommended for prostatitis.

The study's limitations include its single-center design and relatively short duration, which may not represent the entire country. However, as a major referral hospital, its data provide a strong sentinel indicator of the resistance problem in the region. The absence of molecular characterization means the specific resistance mechanisms (e.g., target site mutations in *gyrA/parC*, plasmid-mediated quinolone resistance genes) were not identified.

5. CONCLUSION

The prevalence of resistance to fluoroquinolones among *E. coli* isolates from acute prostatitis patients in Niamey has reached a critical level. This finding strongly indicates that fluoroquinolones should no longer be used for empirical treatment of this condition in this region. Treatment decisions must be guided by local antimicrobial susceptibility data, and urine culture with susceptibility testing is essential for effective patient management. This study highlights the urgent need for robust antimicrobial stewardship and continuous surveillance to preserve the efficacy of remaining antibiotics.

6. RECOMMENDATIONS

Based on the findings of this study, the following recommendations are proposed:

- **Revision of Clinical Guidelines:** Local and national treatment guidelines for acute prostatitis should be immediately revised. Fluoroquinolones should be removed as a first-line empirical therapy option. Alternative agents, guided by local susceptibility data (such as third-generation cephalosporins or fosfomycin, where appropriate), should be considered.
- **Promotion of Culture-Guided Therapy:** Clinicians should be strongly encouraged to obtain urine cultures and susceptibility testing before or during the initiation of treatment for acute prostatitis to allow for targeted, effective therapy and de-escalation.
- **Establishment of Antimicrobial Surveillance:** A systematic and continuous national surveillance program for antimicrobial resistance in uropathogens is urgently needed in Niger. This will enable the tracking of resistance trends and provide data to inform and update treatment guidelines regularly.
- **Implementation of Antibiotic Stewardship Programs:** Health authorities and hospitals must implement strict antibiotic stewardship programs to control the prescription and use of antibiotics, particularly fluoroquinolones, in both community and hospital settings to curb the further development of resistance.
- **Future Research:** Further research, including multicenter studies and molecular characterization of resistance genes (e.g., ESBLs, PMQR genes), is essential to fully understand the epidemiology and mechanisms of resistance in the region.

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