

Bacteriological and Resistance Profile in Isolates from Diabetic Patients

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Abstract

Background: Diabetes mellitus has become a global epidemic illness and poses a threat for development of resistant bacterial infections. **Aim:** This study was aimed to know the bacteriological and resistance profile of isolates obtained from diabetic patients. **Materials and Methods:** The bacterial isolates obtained from various samples of diabetic patients admitted in medicine department in 6-month period were identified and tested for antibiotic susceptibility. The extended spectrum beta-lactamases (ES β L), AmpC, and metallo-beta-lactamases (M β L) enzymes were detected in gram-negative bacilli. Methicillin, macrolide-lincosamide-streptogramin (MLS), and linezolid resistance in *Staphylococcus* spp. were detected. High-level aminoglycoside resistance (HLAR) in *Enterococcus* spp. was also tested. **Results:** In all, 38 of 125 diabetic patients (30.4%) had bacterial infection, 18 patients had wound infections, 18 had urinary tract infections (UTIs), and 2 had respiratory tract infections. *Escherichia coli* among gram-negative bacteria and *Staphylococcus aureus* among gram-positive bacteria were the predominant pathogens. 32.5% gram-negative bacilli were AmpC producers, 37.5% were M β L producers, and 40% were ES β L producers. Methicillin and MLS resistance was found in 50% and 33.3% isolates of *Staphylococcus* spp., respectively. HLAR resistance was alarming in *Enterococcus* spp. Polymyxin among gram-negative bacteria and vancomycin for gram-positive bacteria were the last resort with highest susceptibility rates to treat infections among diabetic patients. **Conclusion:** Resistant bacterial infections in diabetic patients are common. The presence of various resistance mechanisms in isolates of our study shows that therapeutic failure can occur if empirical prescription is unsubstantiated.

Keywords: Antibiotic resistance, Diabetes mellitus, Therapeutic failure

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Introduction

Diabetes mellitus has become a global epidemic illness^[1] and poses a threat for development of resistant bacterial infections. Diabetic patients are more prone to life-threatening infections than nondiabetic patients;^[2-4] therefore, they have more exposure to antibiotics. Diabetic patients have greater problems with healing of infections because of reduced blood supply, which affects the body's ability to fight infection.^[5] When a diabetic patient contracts pneumonia, wound infections, and urinary tract infections, the illness is often more

frequent than in nondiabetic patients.^[2,5] The studies discussing the profile and resistance mechanisms in isolates obtained from diabetic patients are regularly needed to decide empirical therapies in such high-risk patients.

Therefore, this study was attempted to know the bacteriological profile and associated resistance in isolates from diabetic patients.

Materials and Methods

This prospective study was conducted on pus, urine, and sputum/blood samples obtained from consecutive diabetic patients ($n=125$) admitted to Medicine Department of Govt. Medical College and Sushila Tiwari Hospital, Haldwani, Uttarakhand, from January to June 2009. The mean age of diabetic patients was 42.3 years, and sex ratio male:female was 1.4:1. The patients included in the study were those of type 2 diabetes

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mellitus who had poor control of glycemia (assessed on basis of repeated fasting plasma glucose levels), history of infection, hospitalization, and antibiotics exposure (either complete or incomplete course) in last 3 months putting them at increased risk of wound infection, urinary tract infection (UTI), and pneumonia.

Thirty-five pus (25 pedal wounds and 10 nonpedal wounds), 65 urine, and 25 sputum/blood samples were collected and transported within 30 minutes to microbiology laboratory. Pus samples were cultured on blood agar and MacConkey agar plates, blood/sputum on blood and chocolate agar, and urine was cultured on Cysteine lactose electrolyte deficient (CLED) medium as per standard protocol.^[6] The bacterial isolates obtained from various specimens were identified^[6] and tested for antimicrobial susceptibility testing as per our institutional antibiotic policy for both first line and second line of antibiotics (Hi Media Laboratories, India) by Kirby Bauer disc diffusion method.^[7] Multidrug resistance was defined as resistance to three or more groups of drugs.

Wound infection was defined as evidence of microorganism(s) from pus culture.^[8] A diagnosis of pneumonia was made by sputum culture and by isolation of a compatible organism from blood.^[6] Presence of organism(s) of at least 10^5 cfu/mL urine specimens with clinical symptoms indicated UTI.^[6]

Detection of resistance mechanisms by phenotypic methods

ES β L, AmpC, and M β L detection

The gram-negative bacilli leading to resistance for third-generation cephalosporins, cephamycins, and carbapenems on routine screening were also phenotypically tested for detection of extended spectrum beta-lactamase (ES β L), AmpC, and metallo-beta-lactamase (M β L) enzymes, respectively. Briefly, Mueller-Hinton agar plates were prepared and inoculated with standardized inoculums to form a lawn culture. The combined disc methods were used to confirm above resistance mechanisms as described elsewhere.^[9-11] Discs of ceftazidime (30 μ g) and ceftazidime clavulanate (30/10 μ g) for ES β L detection, cefoxitin, and cefoxitin with cloxacillin (30 + 500 μ g, prepared in house) for AmpC and imipenem (30 μ g) and imipenem with Ethylene diaminetetra acetic acid (EDTA) (30 μ g+5 μ g, prepared in house) for M β L detection were used. The increase in inhibition zone of ceftazidime clavulanate disc \geq 5 mm than the ceftazidime disc alone was considered ES β L positive.^[9] If the increase in inhibition zone with cefoxitin and cloxacillin disc was \geq 5 mm than the cefoxitin disc alone, it was considered AmpC positive (slight modification of Ruppé *et al.*).^[10]

The increase in inhibition zone with imipenem and EDTA disc \geq 7 mm than the imipenem disc alone was considered M β L positive.^[11]

Methicillin resistance

The methicillin resistance in *Staphylococcus* spp. was tested by cefoxitin disc (30 μ g) as documented in Clinical and Laboratory Standard Institute (CLSI).^[12] Cefoxitin disc diffusion of \leq 21 mm for *S. aureus* and \leq 24 mm for coagulase negative *Staphylococcus* (CNS) was reported as methicillin resistant.

Linezolid resistance

Agar dilution method was done to confirm linezolid resistance (MIC \geq 8 μ g/ml) in *Staphylococcus* spp.^[13]

High-level aminoglycoside resistance

High content gentamicin disc (120 μ g) was used to detect high-level aminoglycoside resistance (HLAR) in *Enterococcus* spp. The whole zone diameter of \leq 6 mm was considered resistant.^[14]

Macrolide–lincosamide–streptogramin resistance

Staphylococcus spp. with erythromycin resistance (ER-R) were tested for inducible clindamycin resistance using D test keeping 15-mm inter-disc distance as per CLSI recommendation. Briefly, erythromycin (15 μ g) disc was placed at distance of 15 mm (edge to edge) from clindamycin discs (2 μ g) on Mueller-Hinton agar plate previously inoculated with 0.5 McFarland bacterial suspensions. Following overnight incubation at 37°C, flattening of zone around clindamycin in area between the two discs indicated inducible clindamycin resistance^[15]. In our study, two different phenotypes were appreciated after testing as follows:

MLS_B inducible phenotype (MLS_Bi): Isolates resistant to erythromycin while being sensitive to clindamycin and giving D-shaped zone of inhibition around clindamycin with flattening toward erythromycin disc.

MLS_B constitutive phenotype (MLS_Bc): Isolates resistant to both clindamycin and erythromycin.

Results

Of 125 diabetic patients, 38 (30.4%) were infected. The male:female ratio of infections was 25:13 (1.92:1). Of 35 patients, 18 (51.4%) had wound infections, 18 of 65 (27.7%) had UTI, and 2 of 25 (8%) had pneumonia. No patient had concomitant infection.

Sixty-three bacterial species were isolated from 38 samples. Of 63 bacterial isolates, 40 (63.5%) were gram-negative

and 23 (36.5%) were found to be gram-positive. Two bacterial species were isolated from sputum samples ($n=2$; monomicrobial infection), 26 bacterial species from urine samples ($n=18$; 10 were monomicrobial infection while 8 were bimicrobial), while 35 bacterial species were isolated from pus sample ($n=18$; 3 were monomicrobial infections, 13 were bimicrobial, and 2 were polymicrobial with 3 organisms in each sample). Table 1 shows the bacteriological profile of isolates.

The resistance rates of gram-negative bacilli and gram-positive cocci to various antibiotics are shown in Table 2. The various drug-resistance mechanisms investigated among gram-negative and gram-positive bacteria are shown in Table 3.

Discussion

In all, the prevalence of infections in diabetic patients

Table 1: Profile and frequency distribution of gram-positive and gram-negative bacterial isolates obtained from various sites

Bacterial species	Wound ($n=35$)	Urine ($n=26$)	Sputum/blood ($n=2$)	Total ($n=63$)
Gram-negative bacilli ($n=40$)				
<i>Escherichia coli</i>	7	11	-	18 (45)
<i>Klebsiella pneumoniae</i>	4	4	1	9 (22.5)
<i>Pseudomonas aeruginosa</i>	9	2	-	11 (27.5)
<i>Morganella morganii</i>	1	-	-	1 (2.5)
<i>Acinetobacter baumannii</i>	-	1	-	1 (2.5)
Gram-positive cocci ($n=23$)				
<i>Staphylococcus aureus</i>	7	4	1	12 (52.2)
<i>Enterococcus sp.</i>	2	3	-	5 (21.7)
Coagulase negative				
<i>Staphylococcus sp.</i>	5	1	-	6 (26.1)

Table 2: Antibiotic-resistance rates of various bacterial isolates obtained from diabetic patients ($n=63$)

Antibiotics	<i>E. coli</i> ($n=18$)	<i>P. aeruginosa</i> ($n=11$)	<i>K. pneumoniae</i> ($n=9$)	<i>M. morganii</i> ($n=1$)	<i>A. baumannii</i> ($n=1$)	<i>S. aureus</i> ($n=12$)	<i>Enterococcus sp.</i> ($n=5$)	CNS ($n=6$)
Amoxicillin	18 (100)	-	9 (100)	1	1	9 (75)	2 (40)	3 (50)
Ampicillin sulbactam	17 (94.1)	11 (100)	9 (100)	0	1	8 (66.6)	4 (80)	4 (66.6)
Gentamicin	10 (55.5)	6 (54.5)	8 (88.8)	0	1	5 (41.6)	4 (80)	4 (66.6)
Amikacin	3 (16.7)	6 (54.5)	7 (77.7)	0	1	2 (16.7)	5 (100)	3 (50)
Ofloxacin	12 (66.6)	-	5 (55.5)	0	0	7 (58.3)	4 (80)	3 (50)
Cotrimoxazole	14 (77.7)	-	9 (100)	0	0	7 (58.3)	4 (80)	3 (50)
Cefoxitin	10 (55.5)	11 (100)	9 (100)	1	1	-	-	-
Ceftazidime	12 (66.6)	10 (90.9)	8 (88.8)	1	1	-	-	-
Ceftazidime clavulanate	9 (50)	8 (72.7)	8 (88.8)	1	1	-	-	-
Ciprofloxacin	15 (83.3)	9 (81.8)	5 (55.5)	1	0	-	-	-
Chloramphenicol	3 (16.7)	9 (81.8)	4 (44.4)	0	0	-	-	-
Piperacillin tazobactam	5 (27.7)	9 (81.8)	6 (66.6)	0	1	-	-	-
Imipenem	4 (22.2)	6 (54.5)	5 (55.5)	0	0	-	-	-
Polymyxin	1 (5.5)	1 (9.0)	2 (22.2)	0	0	-	-	-
Cefalexin	-	-	-	-	-	5 (41.6)	4 (80)	3 (50)
Cefuroxime	-	-	-	-	-	4 (33.3)	4 (80)	3 (50)
Ciprofloxacin	-	-	-	-	-	10 (83.3)	4 (80)	4 (66.6)
Erythromycin	-	-	-	-	-	5 (41.6)	3 (60)	5 (100)
Azithromycin	-	-	-	-	-	3 (25)	3 (60)	5 (100)
Minocycline	-	-	-	-	-	0	2 (40)	1 (16.6)
Tetracycline	-	-	-	-	-	3 (25)	3 (60)	3 (50)
Linezolid	-	-	-	-	-	0	0	3 (50)
Vancomycin	-	-	-	-	-	0	0	0

was 30.4% with an average of 1.65 organisms per case as compared with nondiabetic population having infection rates of 21.2% with an average 1.2 organisms per case (Singhai, unpublished data). To have further insight, the infection rates and average number of organisms in diabetic and nondiabetic population in various infections have been compared in Table 4. A study on diabetic wound infections has reported 1.52 organism per case,^[16] while another reported 2.3 organism per case;^[17] however, in our study, 1.9 organism per case was found from wound infections. This reinforces the concept of polymicrobial infection in diabetic patients and was seen in our study also.

The number of gram-negative as compared with gram-positive bacterial isolates was high in our study. *E. coli* among gram-negative and *S. aureus* among gram-positive cocci were the most common pathogens. Other studies from India have also reported gram-negative bacilli as predominant pathogen in diabetic infections.^[4,16,17] The anaerobic bacteria also form major pathogens of such infections but could not be assessed because of lack of facilities. Beta-hemolytic *Streptococcus* spp., an important pathogen of wound infections ecology in diabetic patients was not found in our study, which may be attributed to antibiotic usage for infections in past.

The overall susceptibility rates to various antibiotics especially amoxicillin, third-generation cephalosporins, fluoroquinolones tested among gram-negative bacilli and macrolides, amoxycillin, first-generation cephalosporins,

fluoroquinolones among gram-positive cocci were low. The other studies on infections among diabetic patients have also reported high resistance rates to various antibiotics among gram-negative and gram-positive bacteria.^[18-20] Multidrug resistance was found in 82.5% (33/40) among gram-negative and 60.8% (14/23) in gram-positive bacteria.

Another interesting finding of this study was low resistance level to piperacillin/tazobactam and the high resistance level to ceftazidime/clavulanate, which may be attributed either to hyperproduction of AmpC or presence of inhibitor-resistant beta-lactamases (especially in *E. coli*) masking the detection of ESβL by clavulanate but remaining susceptible to inhibition by tazobactam which is in accordance with Bradford *et al.*^[21] When we probed in-depth beta-lactams resistance, 32.5% were AmpC producers, 37.5% were MβL producers, and 40% were ESβL producers among gram-negative bacilli. In our hospital, we have found presence of AmpC, MβL, and ESβL enzymes (28%, 33.5%, and 35%) in gram-negative bacterial strains obtained from clinical specimens of nondiabetic patients slightly low as compared with diabetic patients (Rawat, unpublished data). The focus on detection of the forementioned resistance mechanisms despite of increasing reports on the cephalosporin, cephamycin, and carbapenem-resistance genes from various parts of our country was due to presence of ESβL, AmpC, and MβL in our area as reported by Shahid *et al.*^[22] however, we could not confirm the molecular epidemiology because of lack of molecular setup. The concurrent mechanisms of resistance occurred only in four isolates. Each of the two

Table 3: Detection of various drug-resistance mechanisms in bacteria isolated from diabetic patients

Drug mechanism	Positive*	Negative	Percentage
ESβL	16 (5 K.P, 6 E.C, 5 P.A)	24	40
AmpC	13 (5 K.P, 2 E.C, 5 P.A, 1 A.B)	27	32.5
MβL	15 (5 K.P, 4 E.C, 6 P.A,)	25	37.5
ESβL+AmpC	2 (2 K.P)	-	-
AmpC+MβL	2 (1 E.C, 1 P.A)	-	-
MLS resistance in <i>Staphylococcus aureus</i>	4 (2 MLSBi, 2 MLSBc) [#]	8	33.3
MLS resistance in CNS [‡]	2 (2 MLSBc)	4	33.3
Methicillin-resistance <i>Staphylococcus aureus</i>	6	6	50
Methicillin-resistance CNS	3	3	50
Linezolid-resistance CNS (MIC≥8 µg/ml)	3	3	50
High level aminoglycoside resistance in <i>Enterococcus</i> spp.	3	2	60

*K.P: *Klebsiella pneumoniae*, E.C: *Escherichia coli*, P.A: *Pseudomonas aeruginosa*, A.B: *Acinetobacter baumannii*, [#]MLSBi: Inducible phenotype, : Constitutive phenotype, [‡]CNS: Coagulase negative *Staphylococcus*

Table 4: Infection rates and average number of organisms in diabetic and nondiabetic patients with various infections

Types of infection	Diabetics		Nondiabetics	
	Infection rate (%)	Average number of organisms	Infection rate (%)	Average number of organisms
UTI	27.6	1.4	15.4	1.2
Wound infection	51.4	1.9	48.7	1.3
Pneumonia	8	1	2.5	1

Klebsiella pneumoniae were ESBL and AmpC coproducers, while one *E. coli* and one *Pseudomonas aeruginosa*, each produced AmpC and MBL enzymes simultaneously [Figure 1]. AmpC, ESBL, and MBL producing strains were high in our study population and were in accordance to studies on gram-negative bacilli isolated from diabetic patients.^[23] The major concern is emergence of resistance to third- and fourth-generation of cephalosporins and even more alarming is carbapenem-resistance surfacing nowadays.^[24,25] The carbapenems are often considered a “drug of choice” and are increasingly used in empirical therapy.^[25] However, recent emergence of resistance to this group of antibiotic leaves little options for treating such life-threatening infections as seen in our study group.

The excellent coverage and good oral bioavailability of clindamycin makes it effective therapy against infections in diabetic patients by *Staphylococcus* spp.^[26] However, important issue with clindamycin use is a risk of clinical failure during therapy due to inducible clindamycin resistance.^[26] MLS resistance was found in 33.3% of *S. aureus* and CNS. In our previous study, 38.4% MLS resistance was found in gram-positive cocci.^[15] Failure to identify MLS_Bi may lead to clinical failure of clindamycin therapy, and therefore, its detection is advisable. The true incidence of clindamycin resistance depends on the patient population studied and needs to be assessed to guide the clinicians.^[26] Among *Staphylococcus* spp., methicillin resistance was found in 50% *S. aureus* and 50% CNS, while 50% CNS were linezolid resistant. Methicillin-resistant *Staphylococcus* spp. (MRS) strains with intrinsic resistance to methicillin and all beta-lactam antibiotics are emerging pathogens in hospitalized diabetic patients.^[27] Linezolid, a novel synthetic antibiotic, is considered one of the few effective ways to treat severe methicillin-resistant infections in critically ill patients. Recently, linezolid resistance is reported in our country which may be a consequence of prolonged and injudicious use of this drug^[27,28] and is

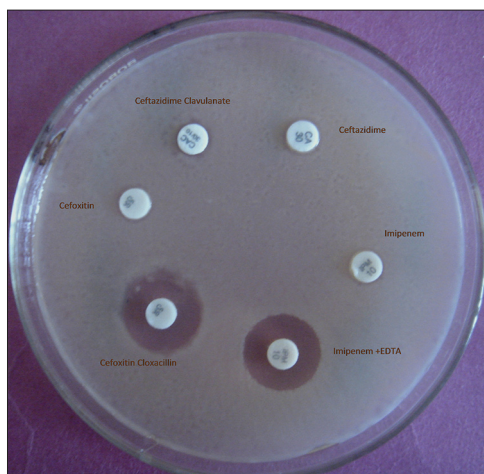


Figure 1: Detection of AmpC and MBL production in *E. coli*

an imperative threat to lose an effective and safe drug for treating MRS. HLAR in *Enterococcus* spp. was also alarming (60%) in our study.

Polymyxin among gram-negative bacilli and vancomycin among gram-positive cocci were the last resorts found to treat multidrug-resistant infections. Our study warrants an urgent need of screening of antibiotic resistance in high-risk population and queries unsubstantiated empirical prescription of antibiotics, which is a common practice in our country.

The detection of various resistance mechanisms by phenotypic methods are easy to interpret, reproducible, and inexpensive and can be included as routine testing protocol. The routine reporting of resistant phenotypes in a target population would allow the clinician to re-evaluate their empirical therapy policies.

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