Bacterial Profile and Antimicrobial Susceptibility of Isolates Recovered from Lower Respiratory Tract Infection for Patients in Rizgary Hospital, Erbil

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Abstract—Recognition of etiologies of lower respiratory tract infection (LRTI) may help in delivering effective treatment options and circumvent emergence of antibiotic resistance. This study is carried out to uncover bacterial profile and antibiotic sensitivity patterns among 310 LRTI patients attended Rizgary Hospital between January 2014 and December 2016. Standard laboratory techniques are applied in collecting, processing, and culturing sputum and bronchial wash specimens. VITEK® 2 compact systems are used to identify bacteria and their antibiotic sensitivity patterns. The results show that *Streptococcus parasanguinis* and *Acinetobacter baumannii* are the most abundant Gram-positive and Gram-negative bacteria (GPB and GNB), respectively, isolated from sputum specimens. From bronchial wash specimens, only GNB are detected and *Serratia marcescens* is the most abundant one. Antibiotic sensitivity tests reveal that *Streptococcus parasanguinis* is the most resistant GPB and *Acinetobacter baumannii* is the most resistant GNB. Sputum recovered GPB are highly resistant to ampicillin, erythromycin, levofloxacin, trimethoprim/ sulfamethoxazole, and tetracycline. Bronchial wash recovered GNB are highly resistant to ampicillin, minocycline, pefloxacin, piperacillin, and ticarcillin. In conclusion, LRTIs are mainly associated with GNB rather than GPB. The recovered *Streptococcus parasanguinis* and *Acinetobacter baumannii* are found to be multidrug resistant pathogens. Ampicillin is ineffective against any of recovered pathogenic bacteria.

Index Terms—*Acinetobacter baumannii*, Ampicillin, Lower respiratory tract infection, Multidrug resistance, *Streptococcus parasanguinis*.

I. Introduction

The lower respiratory tract infections (LRTIs) are common life-threatening illnesses that frequently associated with mortalities worldwide (Madhi and Klugman, 2006; Troeger, et al., 2019). Bacterial-induced LRTI includes bronchitis and pneumonia and increases the risk of pulmonary complications (Kasper, et al., 2006). LRTIs are occurring in children and adult alike, peaking among patients in intensive care units (ICUs). The LRTI acquired in ICUs is best known as hospital-acquired LRTI (Yan, et al., 2018; Karlowsky, et al., 2020). LRTI adverse effects are not limited to population health, but they cause a tangible economic burden on the health-care systems (Ehlken, et al., 2005; Sinha, et al., 2013; Trucchi, et al., 2019).

The majority of bacterial-induced LRTIs are caused by Gram-negative bacteria (GNB), and lower case numbers caused by Gram-positive bacteria (GPB). *Pseudomonas aeruginosa* and *Haemophilus influenzae* (GNB) and *Streptococcus pneumoniae* (GPB) are among the most common bacterial isolates recovered from LTRIs (Kohlenberg, et al., 2008; Khan, et al., 2015). Viruses are also responsible for the development of LRTIs (Ren, et al., 2009; Huang, et al., 2020) and antibiotics are often unnecessarily prescribed for treating these cases that may contribute to the emergence of antibiotic resistance (Pavia, 2011; Shiley, et al., 2015).

Uncovering the etiologies of LRTI has a key role in making the right therapeutic decisions while dealing with this pathological condition (Brookes-Howell, et al., 2012; Langelier, et al., 2018). Unfortunately, in most cases, LRTI treatment is started before culture sensitivity tests are performed (Ali and Butt, 2017). Development of antibiotic resistance may also emerge once patients are given empiric therapy (Yin, et al., 2003; Fatima, et al., 2012; Claeys, et al., 2017). Hence, establishing standard guidelines to deal with LRTIs and their complications are vital to save lives, especially for those who already suffer from antibiotic-resistant bacteria. In more server cases, LRTI patients may suffer from multidrug-resistant pathogens which may make their treatment even more challenging (Woodhead, et al., 2011; Feldman and Richards, 2018).

There are guidelines in place and practiced in a few countries for LRTI management (Christiansen, 1996; Baturin,

et al., 2015; Mahashur, 2018), but these guidelines are not practiced in Iraq. Building up awareness in public and among medical societies is crucial for establishing guidelines based on etiologies explicitly associated with each region. In regard to Erbil city, there are limited data available to represent etiologies of LRTI. Because of the above-stated reasons, and the probability of emergence of new antibiotic resistance cases among the locals in Erbil city (Al-Naqshbandi, et al., 2019), this study was conducted as an attempt to uncover the current status of antibiotic sensitivity patterns of common bacterial isolates collected from LRTIs patients in Erbil city.

II. Materials and Methods

A. Specimen Collection and Transport

The LRT specimens (sputum and bronchial wash) were collected from patients attended Rizgary Teaching Hospital in Erbil city of Kurdistan Region, Iraq, for the period between January 2014 and December 2016. Sputum specimens were collected from patients after educating them to rinse their mouth with water and expectorate with the aid of a deep cough, in the first early morning and gathered directly into a labeled and sterile wide mouth screw cap container. Bronchial wash specimens were aspirated by a pulmonologist in the bronchoscopy unit and collected into a labeled and sterile screw cap container (Mahon, et al., 2014). Sputum and bronchial wash specimens were obtained separately and from different patients. After collection, 310 patients' specimens were transported to laboratories of the Microbiology Department at Rizgary Hospital for analysis.

B. Bacterial Culture and Identification

Good microbiological laboratory practice was applied to deal with sputum or a pellet of centrifuged bronchial wash specimens. Later on, specimens were inoculated separately on blood, chocolate, and MacConkey agar. The inoculum was first smeared thoroughly over the surface of the pre-poured solidified medium. Then, the loop was resterilized and drawn-out from the first site of inoculation into two or three parallel lines on fresh surfaces of the medium. A successive series of strokes were made with the loop that was sterilized between each sequence. At each step, the inoculum was derived from the most distal part of the immediately proceeded strokes. The plates were incubated overnight at 37°C. Number of colonies was counted to calculate bacterial numbers per ml of the specimens. The aerobically incubated bacterial growths were identified based on colony characteristics and outcome of Gram's staining technique (Kumar, 2016). Identification of GPB and GNB genus and species tests were performed by following VITEK® 2 compact system (bioMérieux S.A., France) protocols using the following kits: VITEK®2 GN Reference 21341, VITEK®2 GP Reference 21342, and VITEK®2 AST-GN 82 Reference 413439.

C. Antibiotics

Antimicrobial sensitivity tests were investigated in this study through VITEK® 2 compact system (bioMérieux S.A., France)

kits: VITEK®2 AST-P580 Reference 22233 and VITEK®2 AST-ST01 Reference 410028. Following antibiotics were investigated in this study: AM – Ampicillin, AMC – Amoxicillin/clavulanic acid, AN – Amikacin, ATM – Aztreonam, CAZ – Ceftazidime, CIP – Ciprofloxacin, CM – Clindamycin, CRO – Ceftriaxone, CTX – Cefotaxime, CZ – Cefazolin, E – Erythromycin, ETP – Ertapenem, FA – Fusidic acid, FEP – Cefepime, FOS – Fosfomycin, FT – Nitrofurantoin, GM – Gentamicin, IPM – Imipenem, LEV – Levofloxacin, LNZ – Linezolid, MEM – Meropenem, MNO – Minocycline, MUP – Mupirocin, MXF – Moxifloxacin, OX1 – Oxacillin, P – Benzylpenicillin, PEF – Pefloxacin, PIP – Piperacillin, RA – Rifampicin, SAM – Ampicillin/sulbactam, SXT – Trimethoprim/sulfamethoxazole, TEC – Teicoplanin, TE – Tetracycline, TGC – Tigecycline, TIC – Ticarcillin, TM – Tobramycin, TZP – Piperacillin/tazobactam, and VA – Vancomycin.

D. Data Analysis

Bacterial isolates' abundancy, their distribution, and drug sensitivity were presented in percentage (%). Isolates were considered resistant toward certain antibiotic(s) when the percentile of their resistance was equal or greater than 70%. For the collective antibiotic resistance in GPB and GNB, only the resisted drugs were presented with the value equal or greater than 90%. For these calculations, Microsoft Excel 2010 was used.

TABLE I
DISTRIBUTION OF BACTERIAL GROWTH OF LRTI SPECIMEN'S CULTURE

Bacterial growth	Number of growth (%)	
	Sputum No. (132) (%)	Bronchial wash No. (178) (%)
No growth of bacteria	7 (5.30)	26 (14.61)
Non-pathogenic bacteria	97 (73.49)	124 (69.66)
Pathogenic bacteria	28 (21.21)	28 (15.73)
Gram positive	7 (25)	0 (0)
Gram negative	21 (75)	28 (100)

TABLE II
DISTRIBUTION OF GRAM-POSITIVE AND GRAM-NEGATIVE BACTERIA ISOLATED
FROM LRTI SPECIMEN'S CULTURE

Gram-positive bacterial isolates	Number of bacterial isolates (%)	
	Sputum no. (7) (%)	Bronchial wash no. (0) (%)
Staphylococcus aureus	2 (28.57)	0 (0)
Streptococcus parasanguinis	3 (42.86)	0 (0)
Streptococcus pneumoniae	2 (28.57)	0 (0)
Gram-negative bacterial isolates	Number of bacterial isolates (%)	
	Sputum no. (21)	Bronchial wash no. (28)
Acinetobacter baumannii	7 (33.33)	2 (7.14)
Enterobacter cloacae	1 (4.76)	0 (0)
Escherichia coli	4 (19.05)	4 (14.29)
Klebsiella oxytoca	1 (4.76)	0 (0)
Klebsiella pneumonia	3 (14.29)	5 (17.86)
Proteus mirabilis	1(4.76)	1 (3.57)
Pseudomonas aeruginosa	4 (19.05)	7 (25)
Serratia marcescens	0 (0)	9 (32.14)

TABLE III
THE RESPONSES OF SPUTUM GRAM-POSITIVE ISOLATES TO DIFFERENT ANTIMICROBIAL AGENTS

Agents	*Staphylococcus aureus* (2) (%)	*Streptococcus parasanguinis* (3) (%)	*Streptococcus pneumoniae* (2) (%)
AM – Ampicillin	R2 (100)	R3 (100)	R2 (100)
CM – Clindamycin	R2 (100)	R3 (100)	S1 (50) R1 (50)
CRO – Ceftriaxone	R2 (100)	S2 (66.67) R1 (33.34)	S2 (100)
CTX – Cefotaxime	R2 (100)	S1 (33.34) R2 (66.67)	S2 (100)
E – Erythromycin	R2 (100)	R3 (100)	R2 (100)
FA – Fusidic acid	S2 (100)	R3 (100)	R2 (100)
FOS – Fosfomycin	S2 (100)	R3 (100)	R2 (100)
FT – Nitrofurantoin	S2 (100)	R3 (100)	R2 (100)
GM – Gentamicin	S2 (100)	R3 (100)	R2 (100)
LEV – Levofloxacin	R2 (100)	R3 (100)	R2 (100)
LNZ – Linezolid	S2 (100)	S3 (100)	S2 (100)
MUP – Mupirocin	S2 (100)	R3 (100)	R2 (100)
MXF – Moxifloxacin	S2 (100)	R3 (100)	R2 (100)
OX1 – Oxacillin	S2 (100)	R3 (100)	R2 (100)
P – Benzylpenicillin	R2 (100)	R3 (100)	S2 (100)
RA – Rifampicin	S2 (100)	R3 (100)	R2 (100)
SXT – Trimethoprim/sulfamethoxazole	R2 (100)	R3 (100)	R2 (100)
TE – Tetracycline	R2 (100)	R3 (100)	R2 (100)
TEC – Teicoplanin	S2 (100)	R3 (100)	R2 (100)
TGC – Tigecycline	S2 (100)	R3 (100)	R2 (100)
TM – Tobramycin	S2 (100)	R3 (100)	R2 (100)
VA – Vancomycin	S2 (100)	S3 (100)	S2 (100)

S: Sensitive; R: Resistant

TABLE IV
THE RESPONSES OF SPUTUM GRAM-NEGATIVE ISOLATES TO DIFFERENT ANTIMICROBIAL AGENTS

Agency	*Acinetobacter baumannii* (7) (%)	*Enterobacter cloacae* (1) (%)	*Escherichia coli* (4) (%)	*Klebsiella oxytoca* (1) (%)	*Klebsiella pneumoniae* (3) (%)	*Proteus mirabilis* (1) (%)	*Pseudomonas aeruginosa* (4) (%)
AM – Ampicillin	R7 (100)	R1 (100)	R4 (100)	R1 (100)	R3 (100)	R1 (100)	R4 (100)
AMC – Amoxicillin/clavulanic acid	R7 (100)	R1 (100)	S1 (25) R3 (75)	R1 (100)	R3 (100)	R1 (100)	R4 (100)
AN – Amikacin	R7 (100)	S1 (100)	S2 (50) R2 (50)	S1 (100)	S3 (100)	S1 (100)	R4 (100)
ATM – Aztreonam	R7 (100)	S1 (100)	S3 (75) R1 (25)	S1 (100)	S3 (100)	S1 (100)	R4 (100)
CAZ – Ceftazidime	R7 (100)	S1 (100)	S2 (50) R2 (50)	S1 (100)	S3 (100)	S1 (100)	S1 (25) R3 (75)
CIP – Ciprofloxacin	R7 (100)	S1 (100)	S2 (50) R2 (50)	S1 (100)	S3 (100)	R1 (100)	R4 (100)
CRO – Ceftriaxone	R7 (100)	S1 (100)	S2 (50) R2 (50)	S1 (100)	S2 (66.67) R1 (33.33)	R1 (100)	R4 (100)
CZ – Cefazolin	R7 (100)	R1 (100)	S2 (50) R2 (50)	S1 (100)	S2 (66.67) R1 (33.33)	R1 (100)	R4 (100)
ETP – Ertapenem	R7 (100)	S1 (100)	S2 (50) R2 (50)	S1 (100)	S2 (66.67) R1 (33.33)	R1 (100)	R4 (100)
FEP – Cefepime	R7 (100)	S1 (100)	S3 (75) R1 (25)	S1 (100)	S3 (100)	S1 (100)	R4 (100)
FT – Nitrofurantoin	R7 (100)	R1 (100)	S3 (75) R1 (25)	R1 (100)	R3 (100)	R1 (100)	S1 (25) R3 (75)
GM – Gentamicin	R7 (100)	S1 (100)	S2 (50) R2 (50)	S1 (100)	S3 (100)	S1 (100)	S3 (75) R1 (25)
IPM – Imipenem	R7 (100)	S1 (100)	S2 (50) R2 (50)	S1 (100)	S3 (100)	R1 (100)	R4 (100)
LEV – Levofloxacin	R7 (100)	S1 (100)	S2 (50) R2 (50)	S1 (100)	S2 (66.67) R1 (33.33%)	R1 (100)	R4 (100)
MEM – Meropenem	R7 (100)	S1 (100)	S3 (75) R1 (25)	S1 (100)	S3 (100)	S1 (100)	R4 (100)
MNO – Minocycline	S1 (14.29) R6 (85.71)	R1 (100)	R4 (100)	R1 (100)	S1 (33.33) R2 (66.67%)	R1 (100)	R4 (100)
PEF – Pefloxacin	R7 (100)	R1 (100)	R4 (100)	R1 (100)	S1 (33.33) R2 (66.67%)	R1 (100)	R4 (100)
PIP – Piperacillin	R7 (100)	R1 (100)	R4 (100)	R1 (100)	R3 (100)	S1 (100)	R4 (100)
SAM – Ampicillin/sulbactam	S2 (28.57) R5 (71.43)	R1 (100)	S1 (25) R3 (75)	S1 (100)	S2 (66.67) R1 (33.33)	R1 (100)	R4 (100)
SXT – Trimethoprim/sulfamethoxazole	S2 (28.57) R5 (71.43)	S1 (100)	R4 (100)	S1 (100)	S3 (100)	R1 (100)	R4 (100)
TGC – Tigecycline	S2 (28.57) R5 (71.43)	S1 (100)	S2 (50) R2 (50)	R1 (100)	S2 (66.67) R1 (33.33)	R1 (100)	R4 (100)
TIC – Ticarcillin	R7 (100)	S1 (100)	R4 (100)	R1 (100)	R3 (100)	S1 (100)	R4 (100)
TM – Tobramycin	R7 (100)	R1 (100)	S3 (75) R1 (25)	S1 (100)	S3 (100)	S1 (100)	S1 (25) R3 (75)
TZP – Piperacillin/tazobactam	R7 (100)	S1 (100)	S2 (50) R2 (50)	S1 (100)	S3 (100)	S1 (100)	R4 (100)

S: Sensitive; R: Resistant

III. Results

From different LRTIs patients, 132 sputum and 178 bronchial wash specimens were collected. Out of all sputum specimens, 5.3% showed no bacterial growth, 73.49% showed non-pathogenic bacterial growth, and 21.21% generated pathogenic bacterial growth. Our data revealed that 14.61% of bronchial wash specimens showed no bacterial growth, 69.66% produced non-pathogenic bacteria, and 15.73% generated pathogenic bacteria (Table I). About 25% of sputum pathogenic isolates were GPB (75% were GNB), and bronchial wash isolates were entirely GNB (Tables I and II).

Further analysis of sputum pathogenic isolates showed that *Streptococcus parasanguinis* was the most common GPB and *Acinetobacter baumannii* was the most common GNB. Bronchial wash specimens "entirely" generated GNB, and *Serratia marcescens* was the most detectable species (Table II). Antibiotic susceptibility tests for sputum Gram-positive isolates showed that the most resistant bacteria were *Streptococcus parasanguinis* and the most sensitive one was *Staphylococcus aureus* (Table III). Antibiotic susceptibility test also showed that *Acinetobacter baumannii* was the most resistant sputum Gram-negative isolate, and *Klebsiella pneumoniae* was the most sensitive pathogenic bacteria (Table IV). Antibiotic susceptibility testes for bronchial wash Gram-negative isolates showed that *Acinetobacter baumannii* was the most resistant, and *Klebsiella pneumoniae* was the most sensitive bacteria (Table V).

Antibiotic susceptibility tests also showed that GPB, collectively, were highly resistant to the following antibiotics: Ampicillin, erythromycin, levofloxacin, trimethoprim/sulfamethoxazole, and tetracycline. Our data also showed that all GNB isolated from sputum specimens (except for *Enterobacter cloacae*) have different responses toward studied antibiotics in comparison with the same bacteria that were isolated from bronchial wash specimens (Tables VI and VII).

Staphylococcus aureus recovered from sputum specimens were sensitive to thirteen different types of antibiotics (Table III). *Klebsiella pneumoniae* recovered from sputum specimens was sensitive toward 19 different types of antibiotics (Table IV). *K. pneumoniae* recovered from bronchial wash specimens was sensitive toward 17 different types of antibiotics (Table V).

IV. Discussion

In this study, 310 specimens were collected from patients hospitalized for LRIs treatment and then investigated for bacterial distribution patterns and antibiotic sensitivity. Our data showed no bacterial growths in 5.3% of sputum and 14.61% of bronchial wash specimens (Table I). The undetectable bacterial growth might not reflect the absence of LRIs, as some LRIs cases are due to fungal or viral infections (Troeger, et al., 2019; Barac, et al., 2018). However, this suggestion was not

TABLE V
THE RESPONSES OF BRONCHIAL WASH GRAM-NEGATIVE ISOLATES TO DIFFERENT ANTIMICROBIAL AGENTS

Agency	*Acinetobacter baumannii* (2) (%)	*Escherichia coli* (4) (%)		*Klebsiella pneumoniae* (5) (%)		*Proteus mirabilis* (1) (%)	*Pseudomonas aeruginosa* (7) (%)		*Serratia marcescens* (9) (%)	
AM – Ampicillin	R2 (100)	R4 (100)		R5 (100)		R1 (100)	R7 (100)		R9 (100)	
AMC – Amoxicillin/ clavulanic acid	R2 (100)	S1 (25)	R3 (75)	R5 (100)		S1 (100)	R7 (100)		R9 (100)	
AN – Amikacin	R2 (100)	S3 (75)	R1 (25)	S3 (60)	R2 (40)	R1 (100)	S6 (85.71)	R1 (14.29)	S5 (55.56)	R4 (44.44)
ATM – Aztreonam	R2 (100)	S2 (50)	R2 (50)	S3 (60)	R2 (40)	R1 (100)	R7 (100)		S1 (11.11)	R8 (88.89)
CAZ – Ceftazidime	R2 (100)	S3 (75)	R1 (25)	S3 (60)	R2 (40)	S1 (100)	S7 (100)		S4 (44.44)	R5 (55.56)
CIP – Ciprofloxacin	R2 (100)	S2 (50)	R2 (50)	S3 (60)	R2 (40)	S1 (100)	S5 (71.43)	R2 (28.57)	S5 (55.56)	R4 (44.44)
CRO – Ceftriaxone	R2 (100)	S2 (50)	R2 (50)	S3 (60)	R2 (40)	S1 (100)	R7 (100)		S3 (33.33)	R6 (66.67)
CZ – Cefazolin	R2 (100)	S2 (50)	R2 (50)	S3 (60)	R2 (40)	S1 (100)	R7 (100)		R9 (100)	
ETP – Ertapenem	R2 (100)	S3 (75)	R1 (25)	S3 (60)	R2 (40)	R1 (100)	R7 (100)		S7 (77.78)	R2 (22.22)
FEP – Cefepime	R2 (100)	S3 (75)	R1 (25)	S3 (60)	R2 (40)	S1 (100)	S6 (85.71)	R1 (14.29)	S8 (88.89)	R1 (11.11)
FT – Nitrofurantoin	R2 (100)	S1 (25)	R3 (75)	R5 (100)		S1 (100)	S7 (100)		S4 (44.44)	R5 (55.56)
GM – Gentamicin	R2 (100)	S2 (50)	R2 (50)	S5 (100)		S1 (100)	S6 (85.71)	R1 (14.29)	S4 (44.44)	R5 (55.56)
IPM – Imipenem	R2 (100)	S4 (100)		S3 (60)	R2 (40)	R1 (100)	S4 (57.14)	R3 (42.86)	S7 (77.78)	R2 (22.22)
LEV – Levofloxacin	R2 (100)	S1 (25)	R3 (75)	S3 (60)	R2 (40)	S1 (100)	S6 (85.71)	R1 (14.29)	S7 (77.78)	R2 (22.22)
MEM – Meropenem	R2 (100)	S3 (75)	R1 (25)	S3 (60)	R2 (40)	R1 (100)	S5 (71.43)	R2 (28.57)	S5 (55.56)	R4 (44.44)
MNO – Minocycline	R2 (100)	S1 (25)	R3 (75)	R5 (100)		R1 (100)	S1 (14.29)	R6 (85.71)	S1 (11.11)	R8 (88.89)
PEF – Pefloxacin	R2 (100)	S1 (25)	R3 (75)	R5 (100)		R1 (100)	S1 (14.29)	R6 (85.71)	S1 (11.11)	R8 (88.89)
PIP – Piperacillin	R2 (100)	S1 (25)	R3 (75)	R5 (100)		R1 (100)	S1 (14.29)	R6 (85.71)	S1 (11.11)	R8 (88.89)
SAM – Ampicillin/ sulbactam	R2 (100)	S1 (25)	R3 (75)	S3 (60)	R2 (40)	S1 (100)	R7 (100)		R9 (100)	
SXT – Trimethoprim/ sulfamethoxazole	R2 (100)	S1 (25)	R3 (75)	S3 (60)	R2 (40)	R1 (100)	S6 (85.71)	R1 (14.29)	S4 (44.44)	R5 (55.56)
TGC – Tigecycline	R2 (100)	S2 (50)	R2 (50)	S3 (60)	R2 (40)	R1 (100)	R7 (100)		S4 (44.44)	R5 (55.56)
TIC – Ticarcillin	R2 (100)	S1 (25)	R3 (75)	R5 (100)		R1 (100)	R7 (100)		S1 (11.11)	R8 (88.89)
TM – Tobramycin	R2 (100)	S2 (50)	R2 (50)	S3 (60)	R2 (40)	S1 (100)	S6 (85.71)	R1 (14.29)	S4 (44.44)	R5 (55.56)
TZP – Piperacillin/ tazobactam	R2 (100)	S2 (50)	R2 (50)	S3 (60)	R2 (40)	S1 (100)	S5 (71.43)	R2 (28.57)	S7 (77.78)	R2 (22.22)

S: Sensitive; R: Resistant

conformed since the focus of this study was only on bacteria. On the other hand, there is a possibility that some of the non-bacterial growths are due to anaerobic bacteria (Brook, 1997; Kedzia, et al., 2003), since we did not culture our specimens in anaerobic conditions we cannot confirm that.

Our data also showed that more than 73% of sputum and about 70% of bronchial wash specimens generated non-pathogenic bacterial growth, and this may indicate the presence of normal flora in the collected specimens (Budayanti, et al., 2019).

From 21.21% sputum specimens pathogenic bacteria were recovered (25% GPB and 75% GNB). *Streptococcus parasanguinis* (42.86%) was the most abundant one among GPB, and *Acinetobacter baumannii* (33.33%) was the most abundant one among GNB. Out of all bronchial wash specimens, 15.73% of them generated pathogenic bacteria (100% GNB), and *Serratia marcescens* (32.14%) was the

most abundant species (Table II). In regard to bacterial genus and species, different results were reported in other studies carried out in other countries in the past few years. Khan, et al. (2015) reported that *Pseudomonas aeruginosa* and *Haemophilus influenzae* (GNB) and *Streptococcus pneumoniae* (GPB) were the most detectable bacteria in LTRIs patient. Furthermore, Tchatchouang, et al. (2019) reported in their study that *Streptococcus pneumoniae* and *Haemophilus influenzae* were the most abundant bacteria in LRTI and followed by *Klebsiella pneumoniae* and *Staphylococcus aureus* (Khan, et al., 2015; Tchatchouang, et al., 2019). Since GNB represent more than 87% of all pathogenic bacteria recovered from our specimens, they were the most LRTI associated pathogens, and this came in agreement with other studies (Kohlenberg, et al., 2008; Bali, et al., 2016).

For antibiotic sensitivity tests, our data showing that among GPB recovered from sputum specimens, *Streptococcus*

TABLE VI

PATTERN OF ANTIMICROBIAL RESISTANCE AMONG DETECTED BACTERIA

Isolate type	No. of R.A.	Resisted antibiotics
Sputum Gram-positive isolates		
Streptococcus parasanguinis	18	Ampicillin, Clindamycin, Erythromycin, Fusidic acid, Fosfomycin, Nitrofurantoin, Gentamicin, Levofloxacin, Mupirocin, Moxifloxacin, Oxacillin, Benzylpenicillin, Rifampicin, Trimethoprim/Sulfamethoxazole, Tetracycline, Teicoplanin, Tigecycline, Tobramycin.
Streptococcus pneumoniae	16	Ampicillin, Erythromycin, Fusidic acid, Fosfomycin, Nitrofurantoin, Gentamicin, Levofloxacin, Mupirocin, Moxifloxacin, Oxacillin, Rifampicin, Trimethoprim/Sulfamethoxazole, Tetracycline, Teicoplanin, Tigecycline, Tobramycin.
Staphylococcus aureus	9	Ampicillin, Clindamycin, Ceftriaxone, Cefotaxime, Erythromycin, Levofloxacin, Benzylpenicillin, Trimethoprim/Sulfamethoxazole, Tetracycline.
Sputum Gram-negative isolates		
Acinetobacter baumannii	24	Ampicillin, Amoxicillin/clavulanic acid, Amikacin, Aztreonam, Ceftazidime, Ciprofloxacin, Ceftriaxone, Cefazolin, Ertapenem, Cefepime, Nitrofurantoin, Gentamicin, Imipenem, Levofloxacin, Meropenem, Minocycline, Pefloxacin, Piperacillin, Ampicillin/sulbactam, Trimethoprim/sulfamethoxazole, Tigecycline, Ticarcillin, Tobramycin, Piperacillin/tazobactam.
Pseudomonas aeruginosa	23	Ampicillin, Amoxicillin/clavulanic acid, Amikacin, Aztreonam, Ceftazidime, Ciprofloxacin, Ceftriaxone, Cefazolin, Ertapenem, Cefepime, Nitrofurantoin, Imipenem, Levofloxacin, Meropenem, Minocycline, Pefloxacin, Piperacillin, Ampicillin/sulbactam, Trimethoprim/sulfamethoxazole, Tigecycline, Ticarcillin, Tobramycin, Piperacillin/tazobactam.
Proteus mirabilis	14	Ampicillin, Amoxicillin/clavulanic acid, Ciprofloxacin, Ceftriaxone, Cefazolin, Ertapenem, Nitrofurantoin, Imipenem, Levofloxacin, Minocycline, Pefloxacin, Ampicillin/sulbactam, Trimethoprim/sulfamethoxazole, Tigecycline.
Enterobacter cloacae	9	Ampicillin, Amoxicillin/clavulanic acid, Cefazolin, Nitrofurantoin, Minocycline, Pefloxacin, Piperacillin, Ampicillin/sulbactam, Tobramycin.
Escherichia coli	8	Ampicillin, Amoxicillin/clavulanic acid, Amikacin, Minocycline, Pefloxacin, Piperacillin, Ampicillin/sulbactam, Trimethoprim/sulfamethoxazole.
Klebsiella oxytoca	8	Ampicillin, Amoxicillin/clavulanic acid, Nitrofurantoin, Minocycline, Pefloxacin, Piperacillin, Tigecycline, Ticarcillin.
Klebsiella pneumoniae	5	Ampicillin, Amoxicillin/clavulanic acid, Nitrofurantoin, Piperacillin, Ticarcillin.
Bronchial wash Gram-negative isolates		
Acinetobacter baumannii	24	Ampicillin, Amoxicillin/clavulanic acid, Amikacin, Aztreonam, Ceftazidime, Ciprofloxacin, Ceftriaxone, Cefazolin, Ertapenem, Cefepime, Nitrofurantoin, Gentamicin, Imipenem, Levofloxacin, Meropenem, Minocycline, Pefloxacin, Piperacillin, Ampicillin/sulbactam, Trimethoprim/sulfamethoxazole, Tigecycline, Ticarcillin, Tobramycin, Piperacillin/tazobactam.
Pseudomonas aeruginosa	12	Ampicillin, Amoxicillin/clavulanic acid, Aztreonam, Ceftriaxone, Cefazolin, Ertapenem, Minocycline, Pefloxacin, Piperacillin, Ampicillin/sulbactam, Tigecycline, Ticarcillin.
Proteus mirabilis	12	Ampicillin, Amikacin, Aztreonam, Ertapenem, Imipenem, Meropenem, Minocycline, Pefloxacin, Piperacillin, Trimethoprim/sulfamethoxazole, Tigecycline, Ticarcillin.
Escherichia coli	10	Ampicillin, Amoxicillin/clavulanic acid, Nitrofurantoin, Levofloxacin, Minocycline, Pefloxacin, Piperacillin, Ampicillin/sulbactam, Trimethoprim/sulfamethoxazole, Ticarcillin.
Serratia marcescens	9	Ampicillin, Amoxicillin/clavulanic acid, Aztreonam, Cefazolin, Minocycline, Pefloxacin, Piperacillin, Ampicillin/sulbactam, Ticarcillin.
Klebsiella pneumoniae	7	Ampicillin, Amoxicillin/clavulanic acid, Nitrofurantoin, Minocycline, Pefloxacin, Piperacillin, Ticarcillin.

R.A.: Resisted antibiotic

parasanguinis was the most resistant and *Staphylococcus aureus* was the most sensitive bacteria. For GNB recovered from sputum specimens, *Acinetobacter baumannii* was the most and *Klebsiella pneumoniae* was the least resistant one. Last but not the least, among the isolates recovered from bronchial wash specimens, *Acinetobacter baumannii* was the most resistant and *Klebsiella pneumoniae* was the most sensitive one (Table VI).

Although *Acinetobacter baumannii* that were isolated from two different types of specimens (sputum and bronchial wash), they showed identical resistance toward 24 different types of antibiotics. In contrast to *Acinetobacter baumannii*, *Pseudomonas aeruginosa* that were isolated from bronchial wash specimens were resistant to 12 types of antibiotics in comparison with the same bacteria isolated from sputum specimens that were resistant to 23 types of antibiotics. Other isolates recovered from both types of specimens showed slight differences in this regard, but not in the same patterns that we witnessed in *Acinetobacter baumannii* or *Pseudomonas aeruginosa* (Table VI).

After calculating the collective antibiotic resistance of GPB and GNB, we were able to uncover the most resisted antibiotics. Sputum detected GPB were highly resistant to ampicillin, erythromycin, levofloxacin, trimethoprim/sulfamethoxazole, and tetracycline. Sputum detected GNB were highly resistant to ampicillin and amoxicillin/clavulanic acid. Bronchial wash detected GNB were resistant to ampicillin, minocycline, pefloxacin, piperacillin, and ticarcillin. The highly resisted antibiotic that found to be ineffective against GPB and GNB from both sample types was ampicillin (Table VII). These data suggest the presence of multidrug resistant among LRIs patients in Erbil city. This may emerged as a result of antibiotic abuse, applying non-specific drugs and mismanaged regimen (Prat and Lacoma, 2016).

This study shows the necessity for developing modern and scientifically based protocols by the authorities to deal with LRTI patients and to deliver the most specific and effective antibiotics to slow down life-threatening multidrug resistance problem among the locals of Erbil city. Further investigations are required to assess bacterial profile and antibiotic sensitivity patterns in different time points to uncover the pace of antibiotic resistance among LRTI patients in Erbil city.

V. Conclusion

From this study, we conclude that some of LRTIs are not due to bacterial infections. LRTIs are mainly associated with GNB. This study considers *Streptococcus parasanguinis* and *Acinetobacter baumannii* to be multidrug-resistant pathogens because these two specimens collected bacteria resisted most of the tested antibiotics. The antibiotic drug ampicillin was found to be ineffective in the eradication of all types of recovered pathogenic bacteria. Consequently, there is a pronounced chance of antibiotic resistance problem among LRTI patients. As this study finding can establish a startup for LRTI, further studies are required for better detection of antibiotic resistance pattern development among the locals in Erbil city.

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TABLE VII
Collective antibiotic resistance of gram-positive and -negative bacteria isolated from sputum and bronchial wash specimens.

Isolates	Agent	Resistance (%)
Sputum detected Gram-positive bacteria	Ampicillin	100.0
	Erythromycin	100.0
	Levofloxacin	100.0
	Trimethoprim/sulfamethoxazole	100.0
	Tetracycline	100.0
Sputum detected Gram-negative bacteria	Ampicillin	100.0
	Amoxicillin/clavulanic acid	100.0
Bronchial wash detected Gram-negative bacteria	Ampicillin	100.0
	Minocycline	100.0
	Pefloxacin	100.0
	Piperacillin	100.0
	Ticarcillin	100.0

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