



Epidemiology and microbiological profile comparison between community and hospital acquired infections: A multicenter retrospective study in Lebanon



Roula Matta^a, Souheil Hallit^{a,b,c,d,e,*}, Rabih Hallit^e, Wafaa Bawab^a, Anne-Marie Rogues^f, Pascale Salameh^{a,f,g}

^a Lebanese University, Faculty of Pharmacy, Beirut, Lebanon

^b Occupational Health Environment Research Team, U1219 BPH Bordeaux Population Health Research Center Inserm – Université de Bordeaux, France

^c Psychiatric Hospital of the Cross, Jal Eddib, Lebanon

^d Saint-Joseph University, Faculty of Pharmacy, Beirut, Lebanon

^e Holy Spirit University of Kaslik, Faculty of Medicine and Medical Sciences, Kaslik, Lebanon

^f Unité INSERM 657, Bordeaux 2 University, Bordeaux, France

^g Lebanese University, Faculty of Medicine, Beirut, Lebanon

ARTICLE INFO

Article history:

Received 5 March 2017

Received in revised form 21 August 2017

Accepted 9 September 2017

Keywords:

Hospital
Community
Acquired infections
Resistance

ABSTRACT

Background: The objective of this study is to identify and characterize the species resistance of different pathogens between community acquired and hospital acquired infections pointing at patients' related independent co-morbidities and socio-demographic factors.

Methods: It was a retrospective cohort, multicenter study from five private hospitals located in Beirut and Mount Lebanon. Two hundred fifty-eight adult patients were included.

Results: 110 Gram negative pathogens and 26 Gram positive pathogens were implicated in hospital acquired infections. The Gram-negative bacteria that showed a positive correlation regarding patient's type of infection were *Pseudomonas aeruginosa* (12%), *Klebsiella pneumoniae* (6.2%) and *Acinetobacter baumannii* (3.1%). These bacteria were more frequent in patients with hospital acquired infections ($P = 0.002$, 0.013 and 0.017 respectively). The ratio of methicillin resistant *Staphylococcus aureus*, Extended Spectrum Beta Lactamase producing *Escherichia coli* and *K. pneumoniae* and multi drug *P. aeruginosa* showed high significance in hospital acquired infections. The logistic regression, showed a significant relationship between resistant bacteria and age ($p < 0.001$, ORa = 5.680, CI [2.344; 13.765]) and immunosuppressed state ($p = 0.003$, ORa = 3.137, CI [1.485; 6.630]) and an inverse relationship for Chronic Obstructive Pulmonary Disease (COPD) ($p = 0.006$, ORa = 0.403, CI [0.212; 0.765]).

Conclusion: Our results confirm that hospital acquired infections/bacteria have higher rates of resistance when compared to community acquired; these rates increase with age, immunosuppression and are inversely proportional with COPD. Therefore, physicians should be aware of patients' comorbidities to properly guide initial therapy.

© 2017 The Authors. Published by Elsevier Limited on behalf of King Saud Bin Abdulaziz University for Health Sciences. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Introduction

Hospitals are a threatening environment because of a variable number of virulent pathogens that are brought to it from the community through admitted patients; these patients are then exposed

not only to the indigenous hospital flora but also to the flora of other sick individuals [1]. This is a result of impaired defense mechanism and the colonization of resistant microorganisms [2]. Hospital acquired infections are quite a common feature in the hospitals throughout the world. The prevalence of hospital acquired infections is generally higher in developing countries of limited resources [3]. These bacteria are generally resistant to antibiotics, owing to the heavy use of wide spectrum antibiotics in hospital settings, which puts a high selective pressure on bacteria and induces difficult to treat infections. Thus, hospital acquired infections have

* Corresponding author at: Street 8, building 560, 1st floor, Biakout, Mount Lebanon, Lebanon.

E-mail address: souheilhallit@hotmail.com (S. Hallit).

been recognized for over a century as a critical problem affecting the quality of health and a principal source of adverse healthcare outcomes [4].

Gram-negative bacteria are largely responsible for acquired infections. Multi-drug resistant strains are increasingly isolated in these settings, including carbapenemase-producing *Klebsiella pneumoniae*, *Acinetobacter* and *Pseudomonas aeruginosa* [5]. *Acinetobacter baumannii* has also become one of the most significant antibiotic-resistant bacteria causing hospital acquired infections worldwide [6]. One of the main methods through which Gram negative bacteria develop resistance is carrying genes coding for enzymes, such as beta-lactamases, hydrolyzing and inactivating beta-lactam antibiotics [7], beta-lactams being the most widely used class of antibiotics [8]. A major source of resistance in *Escherichia coli* are plasmid-borne Extended-Spectrum β -Lactamases (ESBL), which are classified as enzymes capable of hydrolyzing most β -lactams such as penicillins, extended-spectrum cephalosporins, and monobactams; ESBLs are not inhibited by β -lactamase inhibitors such as clavulanic acid, sulbactam, and tazobactam [9]. Based on this, community-onset ESBL infections have become an important public health issue [10].

Furthermore, *Staphylococcus aureus* gram-positive cocci can withstand harsh environments for extended periods allowing susceptible individuals to become infected through contact with persistently or transiently colonized people [11]. Thus, these bacteria are the most common hospital acquired pathogens with increased morbidity [12]. Penicillin-resistant strains appeared in hospitalized patients within a short time after the introduction of the antibiotic; methicillin, a β -lactamase-resistant derivative of penicillin, subdivides the species into sensitive and resistant subgroups. Methicillin-resistant *S. aureus* (MRSA) appeared in 1961, one year after methicillin was introduced into clinical use [13].

To our knowledge, no study has ever identified characteristics of bacteria comparing community and hospital settings in Lebanon. This multicenter study was designed to identify and characterize the species of different pathogens between community acquired and hospital acquired infections, pointing on patients' related independent co-morbidities and socio-demographic factors.

Materials and methods

Study design

This was a retrospective cohort, multicenter study from five private hospitals located in Beirut and Mount Lebanon over one year. Among these, three were university hospitals whereas two were non-university hospital. The study duration was 6 months.

Population and data collection

The inclusion criterion was the infection diagnosis according to the Centers for Disease Control (CDC) criteria in an adult patient [14]. Only the first episode of infection in the hospital admission was characterized for each patient. Overall, 258 adult patients were included from the five hospitals in Beirut and Mount Lebanon.

Data were collected through a standardized sheet of patient identification. The record included patient-specific parameters such as demographic data, underlying diseases, and risk factors. The infection variables recorded positive culture, type of germs and antibiotic treatment with presence or absence of multi-resistant bacteria. Hospital acquired infections was defined as a localized or systemic condition that resulted from an adverse reaction due to the presence of infectious agents which occurred 48 h or more after hospital admission and was not incubating at the time of admission. Community acquired infections were defined as an infection

detected within 48 h of hospital admitted patients [15]. Age was classified according to the criteria of Acute Physiology and Chronic Health Evaluation (APACHE) score; for this reason, age was categorized into two subgroups less than 44 years and more than 44 years.

Microbiology data

All participating medical centers were responsible for isolates identification and susceptibility testing. Each laboratory performed susceptibility testing according to their own standardized techniques based on current National Committee for Clinical Laboratory standards [16]. Data collected were primarily qualitative (resistant, intermediate or susceptible). All isolates of *E. coli* and *K. pneumoniae* were tested for extended-spectrum beta-lactamase production. *S. aureus* was tested for methicillin resistance. *P. aeruginosa* was tested for its multiple resistances. *Streptococcus pneumoniae* were tested against penicillin susceptibility.

Comorbidity

The comorbidity of patients in the study included mainly immunosuppression, administration of chemotherapy in the 12 months prior to hospital admission, radiation therapy, administration of steroids for at least 3 months prior to hospital admission, infection with human immunodeficiency virus, chronic liver disease, chronic heart failure, chronic respiratory disease, chronic renal failure, hematological disease, cancer, and diabetes mellitus requiring insulin therapy or oral hypoglycemic agents before the infection.

Data analysis

Statistical evaluation was conducted through bivariate and multivariable risk factor analyses, with the aim of identifying selected bacteria and independent factors associated with the presence of a hospital acquired infection compared to patients with a community acquired infection. Data was entered and analyzed, using Statistical Package for Social Sciences (SPSS) version 22 software. In all analysis, a p-value <0.05 was considered significant. The Chi² test was used for comparing categorical variables between groups; when expected values within cells were <5, Fisher exact test was used. A stepwise forward likelihood ratio multivariable logistic regressions was performed with the type of infection (community hospital acquired versus hospital acquired) as the dependent variable, and the characteristics presenting the lowest p-values in the bivariate analysis as the independent ones. The final model was selected after ensuring models adequacy to data by Hosmer–Lemeshow test.

Results

Population

258 patients were included in this study. Of all patients, 142 (55%) had a hospital acquired infection and 116 (45%) community acquired. Almost half of the patients (50.8%) patients were from community hospitals and (49.2%) from university hospitals.

Correlation between hospital-acquired infection and clinical information

Bivariate analysis was done to assess the relationship between hospital acquired infection and patients' clinical information. Tachycardia was higher in patients with hospital acquired infections compared to the community (83.6% vs 64.1% p<0.001). Moreover, these patients had higher prevalence of tachypnea

Table 1
clinical outcome and bacterial localization in community and hospital acquired infection.

	Community acquired N = 142 (55%)	Hospital acquired N = 116 (45%)	Total N = 258	P-value
Temperature >38.3 °C	74 (52.1%)	72 (62.1%)	146 (56.69%)	0.091
Tachycardia >90 beats/min	91 (64.1%)	97 (83.6%)	188 (72.9%)	<0.001
Tachypnea >20 breath/min	47 (33.1%)	52 (44.8%)	99 (38.4%)	0.047
Leucocytes >12,000/mm ³ /<4000/mm ³	93 (65.5%)	92 (79.3%)	185 (71.7%)	0.01
CRP	53 (60.2%)	37 (86%)	90 (34.9%)	0.003
Sepsis	105 (73.9%)	115 (99.1%)	220 (85.3%)	<0.001
Severe sepsis	56 (39.4%)	92 (79.3%)	148 (57.4%)	<0.001
Defined infection	69 (48.6%)	84 (72.4%)	153 (59.3%)	<0.001
Hospital length of stay >14 days	44 (31.2%)	79 (68.1%)	123 (48%)	<0.001
Bacterial localization	Community acquired	Hospital acquired	Total	P-value
Sputum	25 (17.6%)	26 (22.4%)	51 (19.8%)	0.335
Blood	35 (24.6%)	39 (33.6%)	74 (28.7%)	0.113
Urine	64 (45.1%)	60 (51.7%)	124 (48.1%)	0.287
Ascites fluid	5 (3.5%)	7 (6%)	12 (4.7%)	0.340
Skin and soft tissue	3 (2.1%)	5 (4.3%)	8 (3.1%)	0.256
Catheter	4 (2.8%)	14 (12.1%)	18 (7%)	0.004
Stools	11 (7.7%)	16 (13.8%)	27 (10.5%)	0.114
Bronchoscopy aspiration	4 (2.8%)	19 (16.4%)	23 (8.9%)	<0.001
CSF	0	2 (1.7%)	2 (0.8%)	0.201

(44.8% vs 33.1% $p=0.047$), leukocytosis or leucopenia (79.3% vs 65.5% $p=0.01$), high C-reactive protein (86% vs 50.2% $p=0.003$). Sepsis was more prevalent in patients with hospital acquired infection (99.1% vs 73.9% $p<0.001$). The median of hospital stay was 14 days; we also found a significant difference between the two groups where patients with hospital acquired infections had a higher hospital stay above the median (68.1% vs 31.2% $p<0.001$).

Regarding the characteristics of infections, we had around 60% of patients microbiologically documented infections; this percentage was significantly higher in patients with hospital versus community acquired infections (72.4% vs 48.6% $p<0.001$). Positive culture from the catheter (12.1% vs 2.8% $p=0.004$) and from bronchoscopy (16.4% vs 2.8% $p<0.001$) were more common in hospital-acquired compared to community infections.

Most common bacterial isolates from different specimen types

Most isolates were obtained from the following specimens: 48.1% from urine, 28.7% from blood, 19.8% from sputum, 10.5% from stools, 8.9% from bronchoscopy, 7% from a catheter, 4.7% from ascites fluid, 3.1% from a skin and soft tissues and 0.8% from cerebrospinal fluid. Within the urinary tract infection and blood culture isolates, *E. coli* was the most common bacteria in hospital acquired infection. For sputum, *A. baumannii*, *Enterobacter cloacae*, *E. coli*, *K. pneumoniae* and *P. aeruginosa* were the most common isolates. *Clostridium* species from stools and *E. coli* and *Candida albicans* species from catheter were mainly isolated. Among all types of different specimen collected, bronchoscopy aspirations and catheter were significantly more commonly collected among hospital acquired infections (16.4% vs 2.8%) and (12.1% vs 2.8%) respectively (Table 1).

Descriptive statistics of the infection

Fifty-eight point four percent of the infections were due to one Gram negative bacteria at least, 48% due to one Enterobacteriaceae, 30.7% due to one anaerobic bacteria at least and 26.2% due to one Gram positive bacteria at least. Around 73% of patients had more than one severity criterion and 50.8% had sepsis diagnosis at 48 h or more after hospital admission. The mean ICU length of stay was around 8 days. It is of note that 18.2% of these patients died. The total mortality at hospital discharge was 27.1% (Table 2).

Table 2
Description of the infections.

	N	(%)
At least one Gram positive bacteria	53	26.2%
At least one Gram negative bacteria	118	58.4%
At least one enterobacteriaceae	97	48%
At least one anaerobic bacteria	62	30.7%
More than one severity criteria	187	72.5%
Interval 48 h or more between admission and sepsis	131	50.8%
ICU length of stay (in days)	8.245	±19.6661
ICU mortality discharge	47	18.2%
Total mortality at hospital discharge	71	27.5%

*ICU = Intensive Care Unit.

**ICU length of stay is expressed as mean ± standard deviation.

Most common organisms isolated implicated in hospital acquired infections

A total of 221 isolates from clinical specimens were collected; 171 Gram negative (77.4%) and 50 Gram positive (22.6%) isolates were detected. Among them, 110 (64.3%) of the Gram negative and 26 (52%) of the Gram positive pathogens were implicated in hospital acquired infections. The most common Gram negative bacilli included are *E. coli*, *P. aeruginosa*, *K. pneumoniae*, *E. cloacae* and *A. baumannii* the total of which made up to 53.5% of the total bacilli isolated. A flow-chart was drawn to indicate the most common bacteria involved in the community and hospital acquired infections, with their respective percentages (Fig. 1).

Although *E. coli* was the most frequent isolated microorganism in both groups (24.6% in the community vs 32.8% in the hospital setting), there were no significant differences between community and hospital acquired infections. The Gram-negative bacteria that showed a positive correlation regarding patient's type of infection were *P. aeruginosa* (19% versus 6.3%) ($p=0.002$), *K. pneumoniae* (10.3% versus 2.8%) ($p=0.013$) and *A. baumannii* (6% versus 0.7%) ($p=0.017$); these germs were thus mostly frequent among patients with hospital acquired infections (Table 3). Moreover, the most common Gram positive cocci were *S. aureus* and coagulase negative *Staphylococcus*, which together represented 13.6% from cocci isolated. None of the Gram positive bacterial isolates showed a significant relationship between community and hospital acquired infections (Table 4).

Table 3
Gram negative isolates collected from patients in hospital and community acquired infections.

	Community acquired N = 142 (55%)	Hospital acquired N = 116 (45%)	Total N = 258	P-value
<i>Escherichia coli</i>	35 (24.6%)	38 (32.8%)	73 (28.3%)	0.150
<i>Pseudomonas aeruginosa</i>	9 (6.3%)	22 (19%)	31 (12%)	0.002
<i>Klebsiella pneumoniae</i>	4 (2.8%)	12 (10.3%)	16 (6.2%)	0.013
<i>Enterobacter cloacae</i>	3 (2.1%)	7 (6%)	10 (3.9%)	0.104
<i>Acinetobacter baumannii</i>	1 (0.7%)	7 (6%)	8 (3.1%)	0.017
<i>Proteus mirabilis</i>	2 (1.4%)	5 (4.3%)	7 (2.7%)	0.249
<i>Haemophilus influenzae</i>	3 (2.1%)	3 (2.6%)	6 (2.3%)	0.559
<i>Klebsiella oxytoca</i>	1 (0.7%)	2 (1.7%)	3 (1.2%)	0.447
<i>Serratia spp</i>	1 (0.7%)	1 (0.9%)	2 (0.8%)	0.698
<i>Stenotrophomonas maltophilia</i>	1 (0.7%)	1 (0.9%)	2 (0.8%)	0.698
<i>Morganella morganii</i>	0	2 (1.7%)	2 (0.8%)	0.201
<i>Pseudomonas spp</i>	0	2 (1.7%)	2 (0.8%)	0.201
<i>Clostridium spp</i>	0	2 (1.7%)	2 (0.8%)	0.201
<i>Burkholderia cepacia</i>	0	1 (0.9%)	1 (0.4%)	0.450
<i>Citrobacter freundii</i>	0	1 (0.9%)	1 (0.4%)	0.450
<i>Corynebacterium JK</i>	0	1 (0.9%)	1 (0.4%)	0.450
<i>Enterobacter spp</i>	0	1 (0.9%)	1 (0.4%)	0.450
<i>Haemophilus para- influenzae</i>	0	1 (0.9%)	1 (0.4%)	0.450
<i>Nocardia spp</i>	0	1 (0.9%)	1 (0.4%)	0.450
<i>Salmonella spp</i>	1 (0.7%)	0	1 (0.4%)	0.550

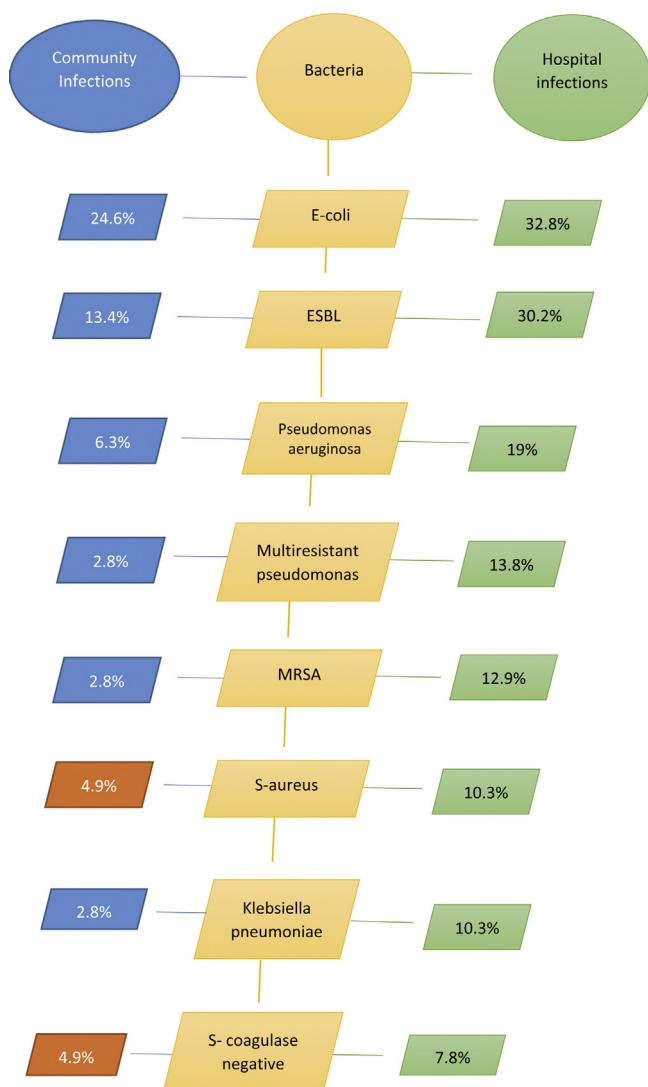


Fig. 1. Flow chart comparing bacteria in community acquired and hospital infections.

Isolates of multi-resistant bacteria

The percentage of MRSA causing hospital acquired infections is 12.9%, compared to 2.8% in community infections ($p=0.001$). ESBL producing *E. coli* and *K. pneumoniae* occurred in 30.2% and 1.7% respectively in hospital acquired infections compared to 13.4% and 0.7% in the community, respectively ($p=0.001$ and 0.424). *P. aeruginosa* showed significance in its resistance to fluoroquinolones ($p<0.001$), imipenem ($p=0.005$), overall multi-resistance ($p=0.001$) and Beta lactams ($p=0.041$) (Table 5).

Factors associated with hospital acquired infections

Bivariate analysis was done to see if there was a relationship between hospital-acquired infection and patient socio-demographic profile and comorbidities. The gender distribution of the patients having hospital acquired infection was 54.2% males and 41.7% females ($p=0.046$). Patients with hospital acquired infection were older than those of the community acquired infected patients in both age groups ($p=0.003$); these patients had higher prevalence of previous comorbidities namely hematologic malignancy (12.1% vs 4.9% $p=0.037$), immunosuppression (24.1% vs 10.6% $p=0.004$), prior administration of steroids (15.5% vs 7.7% $p=0.049$) and chemotherapy (7.8% vs 1.4%). Chronic obstructive pulmonary disease (COPD) was predominant in community patients (16.4% vs 30.3% $p=0.009$) (Table 6).

Table 7 summarizes the results of the forward likelihood logistic regression. There was a significant relationship between older age group (ORa = 5.680; CI [2.344; 13.765] $p<0.001$) and immune-suppressed state (ORa = 3.137; CI [1.485; 6.630] $p=0.003$) with hospital versus community acquired infections, while there was an inverse relationship for COPD (ORa = 0.403 CI [0.212; 0.765] $p=0.006$) (more common among community acquired infections).

Discussion

In this study, patients with hospital acquired infections had higher rates of *P. aeruginosa* and multi-drug resistant *P. aeruginosa*, *K. pneumoniae* and ESBL producing *K. pneumoniae* and *E. coli*, *A. baumannii* and MRSA. Hematologic malignancy, immunosuppression, prior administration of steroids and chemotherapy were risk factors

Table 4
Gram positive isolates collected from patients in hospital and community acquired infections.

	Community acquired N = 142 (55%)	Hospital acquired N = 116 (45%)	Total N = 258	P-value
<i>Staphylococcus aureus</i>	7 (4.9%)	12 (10.3%)	19 (7.4%)	0.098
<i>Staphylococcus coagulase negative</i>	7 (4.9%)	9 (7.8%)	16 (6.2%)	0.349
<i>Enterococcus faecalis</i>	3 (2.1%)	2 (1.7%)	5 (1.9%)	0.595
<i>Pneumococcus spp</i>	4 (2.8%)	0	4 (1.6%)	0.090
<i>Streptococcus agalactiae</i>	1 (0.7%)	3 (2.6%)	4 (1.6%)	0.239
<i>Streptococcus pyogenes</i>	2 (1.4%)	0	2 (0.8%)	0.302

Table 5
Percentage of multiresistant bacteria in hospital and community acquired infections.

Multiresistant bacteria	Community acquired N = 142	Hospital acquired N = 116	P-value
ESBL* gram negative pathogens	19 (13.4%)	35 (30.2%)	0.001
<i>Klebsiella</i> ESBL	1 (0.7%)	2 (1.7%)	0.424
MRSA**	4 (2.8%)	15 (12.9%)	0.002
<i>Pseudomonas</i> Resistant to Imipenem	5 (3.5%)	15 (12.9%)	0.005
<i>Pseudomonas</i> Resistant to Aminoglycosides	3 (2.1%)	8 (6.9%)	0.058
<i>Pseudomonas</i> Resistant to Fluoroquinolones	3 (2.1%)	17 (14.7%)	<0.001
<i>Pseudomonas</i> Resistant to beta-lactams other than imipenem	4 (2.8%)	10 (8.6%)	0.041
Multi-Resistant <i>Pseudomonas</i>	4 (2.8%)	16 (13.8%)	0.001
<i>Streptococcus</i> Resistant to Penicillin	0	1 (0.9%)	0.105

* Extended Spectrum beta lactamase.

** Methicillin resistant *Staphylococcus aureus*.

that showed a positive correlation with hospital acquired infected patients.

COPD was mainly involved in community infected patients. These results are in line with those of other studies. In fact, the Mediterranean region has been identified as an area of hyper-endemicity for multi- drug resistant hospital pathogens [17].

These results are also similar to what was found in both European [18] and non-European countries [19]; as an example, MRSA prevalence varied from less than 1% in Northern Europe to highest than 40% in Southern and Western Europe [18]. With regard to resistant pathogens, (ESBL)-producing *E. coli* was most frequently recovered in hospital acquired patients followed by community acquired infections [20]. Moreover, hospital acquired *P. aeruginosa* were significantly more resistant than community acquired species to all antimicrobial drugs evaluated. These results are consistent with the concept that isolates acquired in the hospital are usually more resistant than those acquired in the community [21], and the majority of infections caused by resistant pathogens were described as hospital acquired [22]. This is in accordance with our results where community-acquired organisms were more susceptible to antimicrobial drugs tested than hospital acquired isolates, but still we found around 13% of ESBL resistance among *E. coli* in the community. In fact, recent data suggest that infections caused by resistant microorganisms are an emerging problem in community patients [20].

Our results showed that *Streptococcus* infections were not frequent in the community setting, in opposite to previous studies [23,24] that showed that this germ was the most common etiological pathogen isolated. This might be due to the fact that currently, *S. pyogenes* is considered a rare cause of community acquired pneumonia, being a clinical entity seen only sporadically after an influenza infection [25]. Furthermore, the proportion of CAP attributed to *S. pneumoniae* showed great variation between countries, with average rates of 24% in Japan, 14% in South Korea and Taiwan, 12% in The Philippines, 8–9% in Thailand, China and India and 4–5% in Malaysia and Singapore [26]. A lack of rigorous microbiological standards, for example, specimen collection following antibiotic use, delays in specimen transport, or culture of specimens with inadequate microscopic screening for white blood

cells, might be expected to reduce the isolation of more fastidious bacteria such as *S. pneumoniae* [27]. We also hypothesize that most of the antibiotic choices are prescribed empirically to patients because of the time required to identify community-acquired respiratory tract infections mainly in people with critical financial conditions, and the time required to find the organism [28].

As for associated factors, we found significant clinical parameters in term of underlying diseases that would distinguish hospital from community acquired infections. Similar to our results, Haley and Schaberg identified age, sex, underlying disease and immunosuppressive therapy as intrinsic factors increasing the risk of infections [29]. In another study, the principal factors that contributed to the risk of acquiring a hospital acquired infection include immunosuppression [30], extensive surgical procedures [31], age and gender [32], and use of prolonged antibiotic therapy [33]; however, in our study, no difference in gender was reported. Neoplastic diseases, such as hematologic malignancies and solid tumors, immune-compromised state [34] were also commonly associated condition in patients with hospital acquired infected patients. Our results were also similar to Kang et al. [35]. Chronic obstructive pulmonary disease had a relationship with community acquired infections due to its exacerbations that usually require hospitalization [36]. Thus, in order to reduce failure of initial antibiotic therapy in patients from the community, it is important to take these factors into account and perform adequate antibiotic empirical control.

Limitations

Our study has limitations. It was an observational, retrospective study conducted on a small sample group with a short duration. Prospective studies involving a large number of patients from different institutions and geographic areas are warranted to confirm our findings and evaluate the need to develop specific guidelines for this new group of patients. Sampling bias is detected by convenient sampling from selected hospitals where university hospitals are over represented [37]; in addition, the season of the year may have a significant influence on rates of diseases and multi drug resistant bacteria [38]; this has to be taken into account in future studies.

Table 6
Bivariate analysis of the predictive factors of hospital acquired infection.

Patients' risk factors	Community acquired 142 (55%)	Hospital acquired 116 (45%)	P-value
Age			
<44 years	30 (21.9%)	107 (78.1%)	0.001
>44 years	8 (7%)	107 (93%)	
Gender			
Male	65 (45.8%)	77 (54.2%)	0.046
Female	67 (58.3%)	48 (41.7%)	
Obesity			
No	79 (57.7%)	58 (42.3%)	0.562
Yes	27 (52.9%)	24 (41.7%)	
Hematologic malignancies			
No	135 (95.1%)	7 (4.9%)	0.037
Yes	102 (87.9%)	14 (12.1%)	
AIDS			
No	142 (100%)	0	0.450
Yes	115 (99.1%)	1 (0.9%)	
Cirrhosis			
No	139 (97.9%)	3 (2.1%)	0.473
Yes	111 (95.7%)	8 (4.3%)	
Chronic heart failure			
No	98 (69%)	44 (31%)	0.365
Yes	86 (74.1%)	30 (25.9%)	
Metastatic cancer			
No	127 (89%)	15 (10.6%)	0.774
Yes	105 (90.5%)	11 (9.5%)	
COPD			
No	99 (69.7%)	43 (30.3%)	0.009
Yes	97 (83.6%)	19 (16.4%)	
Alcoholism			
No	139 (97.9%)	3 (2.1%)	0.183
Yes	110 (94.8%)	6 (5.2%)	
Diabetes mellitus			
No	92 (64.8%)	50 (35.2%)	0.392
Yes	81 (69.8%)	35 (30.2%)	
Chronic renal failure			
No	116 (81.7%)	26 (18.3%)	0.757
Yes	93 (80.2%)	23 (19.8%)	
Osteo-muscular problem			
No	126 (88.7%)	16 (11.3%)	0.482
Yes	106 (91.4%)	10 (8.6%)	
Bone marrow transplantation			
No	141 (99.3%)	1 (0.7%)	0.482
Yes	114 (98.3%)	2 (1.7%)	
Immunocompromised state			
No	127 (89.4%)	15 (10.6%)	0.004
Yes	88 (75.9%)	28 (24.1%)	
Prior administration of prednisone			
No	131 (92.3%)	11 (7.7%)	0.049
Yes	98 (8.5%)	18 (15.5%)	
Chemotherapy			
No	140 (98.6%)	2 (1.4%)	0.002
Yes	107 (92.2%)	9 (7.8%)	
Severe malnutrition			
No	138 (97.2%)	4 (2.8%)	0.735
Yes	111 (95.7%)	5 (4.3%)	
Solid organ transplantation			
No	132 (93%)	10 (7%)	0.746
Yes	109 (94%)	7 (6%)	

Table 7
Logistic regression of predictors of hospital versus community acquired infections.

Independent variable	OR _a	95%CI	P-value
Older age class	5.680	[2.344;13.765]	<0.001
Immuno-compromised state	3.137	[1.485;6.630]	0.003
COPD	0.403	[0.212;0.765]	0.006

Overall percentage: 62.7%; Omnibus test significant; Hosmer Lemeshow $P=0.709$ (Not significant).

Variables initially entered: Age class, COPD, immuno-compromised state, Hematologic malignancy and radiation or chemotherapy.

Conclusion

Our results confirm that hospital acquired infections have higher rates of resistant bacteria when compared to community acquired infections. Microbiologists have to play an important role in the prevention of hospital acquired infections because they are the first to detect the patients' bacteriological flora and their pattern of resistance. Physicians should also be aware of patients' comorbidities to properly guide the initial therapy, particularly age, immunosuppression, and COPD; since resistance is attributed to local epidemiology and uncontrolled use of antimicrobial agents, further research involving a large number of patients from different hospitals are needed in order to confirm our findings and highlight the need for antimicrobial stewardship.

Ethical approval

The Lebanese University ethical committee waived approval of the study since it is an observational non-invasive study that respects participants' autonomy and anonymity; the study followed principles of the Declaration of Helsinki for such types of studies.

Conflicts of interest

None declared.

Funding sources

No funding sources.

References

- [1] Wu CJ, Lee HC, Lee NY, Shih HI, Ko NY, Wang LR, et al. Predominance of Gram-negative bacilli and increasing antimicrobial resistance in nosocomial bloodstream infections at a university hospital in southern Taiwan, 1996–2003. *J Microbiol Immunol Infect* 2006;39(April (2)):135–43.
- [2] Chen Y, Xu X, Liang J, Lin H. Relationship between climate conditions and nosocomial infection rates. *Afr Health Sci* 2013;13(2):339–43.
- [3] Alp E, Leblebicioglu H, Doganay M, Voss A. Infection control practice in countries with limited resources. *Ann Clin Microbiol Antimicrob* 2011;10:36.
- [4] Cornejo-Juárez P, Vilar-Compte D, Peírez-Jimeínez C, N'amendys-Silva SA, Sandoval-Hernández S, Volkow-Fernández P. The impact of hospital-acquired infections with multidrug-resistant bacteria in an oncology intensive care unit. *Int J Infect Dis* 2015;31:31–4.
- [5] Doyle JS, Buising KL, Thursky KA, Worth LJ, Richards MJ. Epidemiology of infections acquired in intensive care units. *Semin Respir Crit Care Med* 2011;32(2):115–38.
- [6] Brusselaers N, Vogelaers D, Blot S. The rising problem of antimicrobial resistance in the intensive care unit. *Ann Intensive Care* 2011;1:47.
- [7] Ho J, Tambyah PA, Paterson DL. Multiresistant Gram-negative infections: a global perspective. *Curr Opin Infect Dis* 2010;23(December (6)):546–53.
- [8] Yao JD, Moellering R. In: Murray PR, Baron EJ, Jorgensen J, Pfaller MA, Tenover FC, Tenover FC, editors. *Manual of clinical microbiology*. 8th ed. Washington, DC, USA: ASM Press; 2003.
- [9] Rogers B, Sidjabat HE, Paterson DL. *Escherichia coli* O25b-ST131: a pandemic, multiresistant, community-associated strain. *J Antimicrob Chemother* 2011;66(January (1)):1–14.
- [10] Pitout JDD. Extraintestinal pathogenic *Escherichia coli*: a combination of virulence with antibiotic resistance. *Front Microbiol* 2012;3(January):9.
- [11] Ben-David D, Mermel LA, Parenteau S. Methicillin-resistant *Staphylococcus aureus* transmission: the possible importance of unrecognized health care worker carriage. *Am J Infect Control* 2008;36(March (2)):93–7.
- [12] Nabera CK. *Staphylococcus aureus* bacteremia: epidemiology, pathophysiology, and management strategies. *Clin Infect Dis* 2009;48(Suppl. 4):S231–7.
- [13] Miall LS, McGinley NT, Brownlee KG, Conway SP. Methicillin resistant *Staphylococcus aureus* (MRSA) infection in cystic fibrosis. *Arch Dis Child* 2001;84:160–2.
- [14] Horan TC, Andrus M, Dudeck MA. CDC/NHSN surveillance definition of health care-associated infection and criteria for specific types of infections in the acute care setting. *Am J Infect Control* 2008;36:309–32.
- [15] Ducloux G, Fabry J, Nicolle L. Prevention of hospital-acquired infections: a practical guide. 2nd ed. WHO/CDS/CSR/EPH/2002.12; 2002.
- [16] National Committee for Clinical Laboratory Standards. Performance standards for antimicrobial disk susceptibility tests: approved standard M2–A6. Wayne, PA, USA: NCCLS; 1997.

- [17] Gur D, Unal S. Resistance to antimicrobial agents in Mediterranean countries. *Int J Antimicrob Agents* 2001;17:21–6.
- [18] Tiemersma EW, Bronzwaer SL, Lyytikäinen O, Degener JE, Schrijnemakers P, Bruinsma N, et al. Methicillin-resistant *Staphylococcus aureus* in Europe, 1999–2002. *Emerg Infect Dis* 2004;10:1627–34.
- [19] Borg MA, de Kraker M, Scicluna E, van de Sande-Bruinsma N, Tiemersma E, Monen J, et al. Prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA) in invasive isolates from southern and eastern Mediterranean countries. *J Antimicrob Chemother* 2007;60:1310–5.
- [20] Son JS, Song JH, Ko KS, Yeom JS, Ki HK, Kim SW, et al. Bloodstream infections and clinical significance of health care associated bacteremia: a multicenter surveillance study in Korean hospitals. *J Korean Med Sci* 2010;25:992–8.
- [21] Guembe M, Cercenado E, Marin M, Insa R, Bouza E. Evolution of antimicrobial susceptibility patterns of aerobic and facultative gram-negative bacilli causing intra-abdominal infections: results from the SMART studies 2003–2007. *Rev Esp Quimioter* 2008;21(3):166–73.
- [22] Zaman R, Dibb WL. Methicillin resistant *Staphylococcus aureus* isolated in Saudi Arabia: epidemiology and antimicrobial resistance patterns. *J Hosp Infect* 1994;26(4):297–300.
- [23] Menon RU, George AP, Menon UK. Etiology and anti-microbial sensitivity of organisms causing community acquired pneumonia: a single hospital study. *J Fam Med Prim Care* 2013;2(July (3)):244.
- [24] Bansal S, Kashyap S, Pal LS, Goel A. Clinical and bacteriological profile of community acquired pneumonia in Shimla, Himachal Pradesh. *Indian J Chest Dis Allied Sci* 2004;46(March (1)):17–22.
- [25] Musher DM, Thorner AR. Community-acquired pneumonia. *N Engl J Med* 2014;371(October (17)):1619–28.
- [26] Peto L, Nadjim B, Horby P, Ngan TT, van Doorn R, Kinh NV, et al. The bacterial aetiology of adult community-acquired pneumonia in Asia: a systematic review. *Trans R Soc Trop Med Hyg* 2014;108(April (6)):326–37.
- [27] Bartlett JG. Diagnostic tests for agents of community-acquired pneumonia. *Clin Infect Dis* 2011;52(May (Suppl. 4)):S296–304.
- [28] Daoud Z, Kourani M, Saab R, Nader MA, Hajjar M. Resistance of *Streptococcus pneumoniae* isolated from Lebanese patients between 2005 and 2009. *Rev Esp Quimioter* 2011;24(2):84–90.
- [29] Haley RW, Schaberg DR. Prevalence and importance of hospital acquired infection. *Am J Med* 1981;70:51. Cited in *Principles of Bacteriology, Virology and Immunity*, Ed. By Topley & Wilson, 8th Ed. 1990, 3: 142.
- [30] Klastersky J. Infections in compromised hosts: considerations on prevention. *Eur J Cancer Clin Oncol* 1989;25(Suppl. 2):S53–61.
- [31] Brett W, Masters PJ, Lang SD, Ikram RB, Hatch SH, Gordon MS. Antibiotic susceptibility of *Streptococcus pneumoniae* in New Zealand. *N Z Med J* 1999;74(8):112–83.
- [32] Barbut F, Petit JC. Epidemiology of *Clostridium difficile*-associated infections. *Clin Microbiol Infect* 2001;7(8):405–10.
- [33] Yassin SF, Young-Fadok TM, Zein NN, Pardi DS. *Clostridium difficile*-associated diarrhea and colitis. *Mayo Clin Proc* 2001;76(7):725–30.
- [34] Cardoso T, Ribeiro O, Aragao I, Costa-Pereira A, Sarmento A. Differences in microbiological profile between community-acquired, healthcare-associated and hospital-acquired infections. *Acta Med Port* 2013;26(July–August (4)):377–84.
- [35] Kang CI, Kim SH, Bang JW, Kim HB, Kim NJ, Kim EC, et al. Community-acquired versus nosocomial *Klebsiella pneumoniae* bacteremia: clinical features, treatment outcomes, and clinical implication of antimicrobial resistance. *J Korean Med Sci* 2006;21:816–22.
- [36] Au DH, Bryson CL, Chien JW, Sun H, Udrys EM, Evans LE, et al. The effects of smoking cessation on the risk of chronic obstructive pulmonary disease exacerbations. *J Gen Intern Med* 2009;24(4):457–63.
- [37] Laupland KB, Ross T, Pitout JD, Church DL, Gregson DB. Investigation of sources of potential bias in laboratory surveillance for anti-microbial resistance. *Clin Invest Med* 2007;30:E159–66.
- [38] Rossello-Urgell J, Vaque-Rafart J, Armadans-Gil LL, Vaquero-Puerta JL, Elorza-Ricart JM, Quintas-Fernández JC, et al. The importance of the day of the week and duration of data collection in prevalence surveys of nosocomial infections. *J Hosp Infect* 2004;57:132–138.