Biomedical Research & Therapy

DOI: 10.15419/bmrat.v5i8.467

Research

Article History:
Received: 08 July 2018
Accepted: 01 August 2018
Published: 24 August 2018

Keywords:
entB, iutA, K2, kfu, Klebsiella pneumoniae, magA, mrkD, rmpA, ybtS

Author for correspondence:
Rashid Ramazanzadeh
e-mail: atrop_t51@yahoo.com

Molecular detection of virulence genes in *Klebsiella Pneumoniae* clinical isolates from Kurdistan Province, Iran

Pegah Shakib[1,2], Morovvat Taheri Kalani[3], Rashid Ramazanzadeh[1,2,4], Amjad Ahmadi[1,2] and Samaneh Rouhi[1,2]

[1]Cellular and Molecular Research Center, Kurdistan University of Medical Sciences, Sanandaj, Iran
[2]Department of Microbiology, School of Medicine, Kurdistan University of Medical Sciences, Sanandaj, Iran
[3]Social Determinants of Health Research Center, Lorestan University of Medical Sciences, Khorramabad, Iran
[4]Iran National Science Foundation (INSF)

Abstract

Introduction: The purpose of this study was to detect *ybtS, entB, mrkD, magA, kfu, iutA, rmpA* and *K2* genes in extended-spectrum beta-lactamase (ESBL) - and non-ESBL producing *Klebsiella pneumoniae*. **Methods**: To this end, 70 *K. pneumoniae* isolates were selected from hospitals of Kurdistan Province, Iran. The ESBL phenotype was conducted utilizing the disc diffusion technique in accordance with CLSI procedures. Detection of virulence factor genes was performed by the PCR in the ESBL and non-ESBL isolates. **Results**: Sixty-two (88.6%) isolates of *K. pneumoniae* were ESBL-producers. Further, *entB* had the most frequency in all the isolates. There were no significant differences between ESBL production and the presence of *ybt S, entB, mrkD, magA, kfu, iutA, rmpA and K2* genes and the presence of these genes and variables such as presence of sex, clinical specimen type, and hvKP phenotype among the ESBL and non-ESBL *K. pneumoniae* isolates. **Conclusion**: In conclusion, in other studies, *K. pneumoniae* strains were separated from liver abscesses and the magA gene was frequently present; however, in our study, the *K. pneumoniae* strains were separated from various clinical specimens and the *magA* gene had low frequency.

1. Background

Klebsiella pneumoniae is a prominent opportunistic pathogen which causes upper respiratory tract infection, diarrhea, pneumonia, urinary tract infection (UTI), and septicemia [1–3]. The prevalence of drug resistance in *K. pneumoniae* has increased, which is because of extended-spectrum beta-lactamase (ESBL) enzymes and appearance of multi-drug resistant (MDR) *K. pneumoniae* [4,5]. In addition, *K. pneumoniae* possesses different virulence factors that contribute to its pathogenicity including lipopolysaccharide (LPS) O-side chain (endotoxin), capsular polysaccharide, adhesions and sidrophores [4,6,7]. The LPS contains lipid A, core, and O-polysaccharide antigen [8]. Capsule polysaccharide (CPS) is a major factor for virulence of *K. pneumoniae* and classified into 77 serological types (K) [9]. Capsular layers engulf the surface of bacteria and prevent bacteria phagocytosis. *K1* and *K2* capsular antigens are the most important ones [10].

Genome of the *K. pneumoniae* capsule comprises gene clusters *cps* (capsular polysaccharide synthesis), *magA* (mucoviscosity associated gene A), *rmpA* and wb (O-specific polysaccharide directed by the wb gene cluster) [11]. *MagA* (35-Kbp) was identified as a K1-specific capsular polymerase gene which acts as a trans-acting activator for biosynthesis of cps. Moreover, *magA* is homologous with the genes involved in glycosylation, transfer and biosynthesis of the LPS [12]. In 2004, *magA* was determined as the major virulence factor of *K. pneumoniae* [2]. It has been reported that *rmpA* can magnify the colony mucoidy of different serotypes of *K. pneumoniae* and act as a plasmid-mediated regulator of extra capsular polysaccharide synthesis [13]. Adhesives include Pilli, the building of protein, and the attachment of bacteria to the host. *MrkD* gene mediates binding to the extracellular matrix, and also codes type 3 fimbria adhesion [14]. *K. pneumoniae* by different siderophores (iron-bound) including enterobactin, yersiniabactin and hydroxamate siderophore obtain iron from transferrin and lactoferrin in host transport proteins. *EntB, ybtS, kfu* and *iutA* genes encode enterobactin, yersiniabactin, iron-uptake system and hydroxamate siderophore [15]. The main purpose of the current study was to detect *ybtS, entB, mrkD, magA, kfu, iutA, rmpA*, and *K2* genes in ESBL and non-ESBL producing *K. pneumoniae* isolated from clinical specimens in Kurdistan Province, Iran.

2. Methods

(a) Identification of bacterial strains

Seventy *K. pneumoniae* isolates were taken from specimens including urine, blood, tracheal aspirates and wound from October 2015 to July 2016 from general hospitals of Kurdistan Province, Iran. All the isolates were cultured on blood and MacConkey agar (Merck, Germany). Colonies were identified by Gram stain and biochemical tests such as urea hydrolysis, H2S production, lysine decarboxylase, lactose fermentation, indole, methyl red, voges proskauer, citrate (IMViC) and oxidase tests [16].

(b) Phenotypic detection of ESBLs strains

Detection of ESBLs was tested by the combination disk diffusion test (CDDT) for the *K. pneumoniae* isolates. The CDDT was performed by ceftazidime (30 μg), cefotaxime (30 μg), cefepime (30 μg), cefpodoxime (30 μg), cefotaxime-clavulanic acid (30/10μg), cefotaxime-clavulanic acid (30/10 μg), cefepime-clavulanic acid (30/10 μg) and cefpodoxime-clavulanic acid (30/10 μg) (Roscoe, Denmark) (Ho *et al.*, 1998). *Escherichia coli* ATCC 25922 and *K. pneumoniae* ATCC 7881 were utilized for negative and positive controls, respectively.

(c) Determination of hypermucoviscosity *K. pneumoniae* (hv-KP) phenotype

Seventy *K. pneumoniae* isolates were separated from the clinical samples and cultivated on blood agar medium (Merck, Germany); then, they were incubated at 37°C for 24 h. Subsequently, the

hypermucoviscosity of *K. pneumoniae* (hv-KP) phenotype was determined by forming a viscous string more than 5 in standard bacteriological loops [3].

(d) Virulence genes identification by PCR amplification

The *K. pneumoniae* isolates were cultured on Luria broth (LB) medium overnight. Then, DNA samples were extracted using Genomic DNA Extraction Kit (SinaClon, Iran). Gene coding virulence factors were detected by the PCR method. PCRs were carried out by using the thermo cycler system (Bio-Rad, Australia) and master mix PCR (YT1553, Iran) and primers were designed by Compain *et al.* (**Table 1**) [14]. Amplification was carried out as follows: initial denaturation at 95°C for 15 min followed by 30 cycles of denaturation at 94°C for 30 s, 60°C for 90 s, and 72°C for 60 s and elongation at 72°C for 10 min. One multiplex PCR was not performed for detection of genes due to unavailability of one control isolate with multiple genes. Control positive isolates were obtained from the Lorestan University of Medical Sciences, Iran.

Table 1. Characteristics of the primers used in PCRs

Primer	Name DNA sequence (5 to 3)	Amplicon size (bp)
ybtS_for	GACGGAAACAGCACGGTAAA	242
ybtS_rev	GAGCATAATAAGGCGAAAGA	
mrkD_for	AAGCTATCGCTGTACTTCCGGCA	340
mrkD_rev	GGCGTTGGCGCTCAGATAGG	
entB_for	GTCAACTGGGCCTTTGAGCCGTC	400
entB_rev	TATGGGCGTAAACGCCGGTGAT	
rmpA_for	CATAAGAGTATTGGTTGACAG	461
rmpA_rev	CTTGCATGAGCCATCTTTCA	
K2_for	CAACCATGGTGGTCGATTAG	531
K2_rev	TGGTAGCCATATCCCTTTGG	
kfu_for	GGCCTTTGTCCAGAGCTACG	638
kfu_rev	GGGTCTGGCGCAGAGTATGC	
iutA_for	GGGAAAGGCTTCTCTGCCAT	920
iutA_rev	TTATTCGCCACCACGCTCTT	
magA_for	GGTGCTCTTTACATCATTGC	1283
magA_rev	GCAATGGCCATTTGCGTTAG	

(e) Statistical analysis

The association between the ESBL production, clinical specimen type, sex ,and hvKP phenotype and presence of *ybtS, entB, mrkD, magA, kfu, iutA, rmpA and K2* genes among the ESBL and non-ESBL *K.pneumoniae* isolates was analyzed by Fisher tests with STATA software program v12.

3. Results

(a) Bacterial isolates

Out of the 70 *K. pneumoniae* isolates, 37 (52.9%), 32 (45.7 %) and 1 isolates (1.4%) were collected from women, men and the hospital environment. Moreover, 50 isolates (71.4%) were obtained from urine, 8 isolates (11.4%) from blood, 10 isolates (14.3%) from tracheal aspirates, 1 isolate (1.4%) from wound, and 1 isolate (1.4%) from the environment (**Table 2**).

Table 2. The characteristics of the hypermucoviscosity clinical isolates

Sample No	Origin	Sex	ESBL production	Virulence genes
Kp6	urine	male	positive	ybt S,entB, mrkD
Kp12	urine	female	positive	ybt S,entB, mrkD
Kp16	urine	male	positive	entB, mrkD, kfu
Kp32	urine	female	positive	ybt S,entB, mrkD, magA
Kp39	tracheal	male	positive	entB, mrkD
Kp43	urine	female	positive	ybt S,entB, mrkD, rmpA
Kp46	urine	female	positive	ybt S,entB, mrkD, rmpA
Kp53	urine	female	positive	ybt S,entB, mrkD
Kp58	urine	female	positive	entB, iutA
Kp60	urine	female	positive	entB

(b) Screening for ESBLs and results hypermucoviscosity K. pneumoniae (hv-KP) strains

The results of screening for the ESBL showed that 62 isolates (88.6%) were ESBL-producing *K. pneumoniae* isolates. Of the 70 clinical isolates, 10 isolates (14.3%) were positive and 60 isolates (85.7%) were negative for the hv-KP test. Table 2 shows characteristics of hypermucoviscosity clinical isolates.

(c) Virulence genes identification

According to the results of screening virulence genes, *entB* (n= 57, 81.4%) was the most prevalent among all the clinical isolates, followed by the *mrkD* (n=46, 65.7 %%), *ybtS* (n=42, 60%), *iutA* (n=8, 11.4%), *kfu* (n=8, 11.4%), *rmpA* (n=4, 5.7%), *magA* (n=1, 1.43%), and *K2* was not detected in any of the isolates. The presence of the *ybtS, entB, mrkD, magA, kfu, iutA, rmpA and K2* genes coding virulence factors was detected among the ESBL and non-ESBL *K. pneumoniae* isolates (**Table 3**).

Table 3. Frequency of virulence factor genes among the ESBL and Non -ESBL *K. pneumoniae* isolates

	Total isolates	ESBL	Non-ESBL	P-Value
ybt S	42(60%)	38(90.5%)	4(9.5%)	0.705
entB	57(81.4%)	52(91.2%)	5(8.8%)	0.161
mrkD	46(65.7%)	43(93.5%)	3(6.5%)	0.113
magA	1(1.4%)	1(100%)	0(0%)	1.000
iutA	8(11.4%)	6(75%)	2(25%)	0.225
Kfu	8(11.4%)	7(87.5%)	1(12.5 %)	1.000
rmpA	4(5.7%)	4(100%)	0(0%)	1.000
K2	0(0 %)	0(0%)	0(0 %)	0.231

4. Discussion

Generally, one of the classes of antibiotics used for treating *K. pneumonia*e is beta-lactams such as cephalosporin [17]. However, the presence of ESBL enzymes impairs the performance of these antibiotics [18]. The difference in sensitivity and drug resistance in different geographic regions can be associated with different patterns of antibiotic use in different areas [19]. In this survey, 88.6% of the clinical isolates were the ESBL producers. Moreover, as shown in the study by Ghasemi, 60% of *K. pneumoniae* isolates were ESBL producers in Shiraz, Iran [20]. Jaskulski *et*

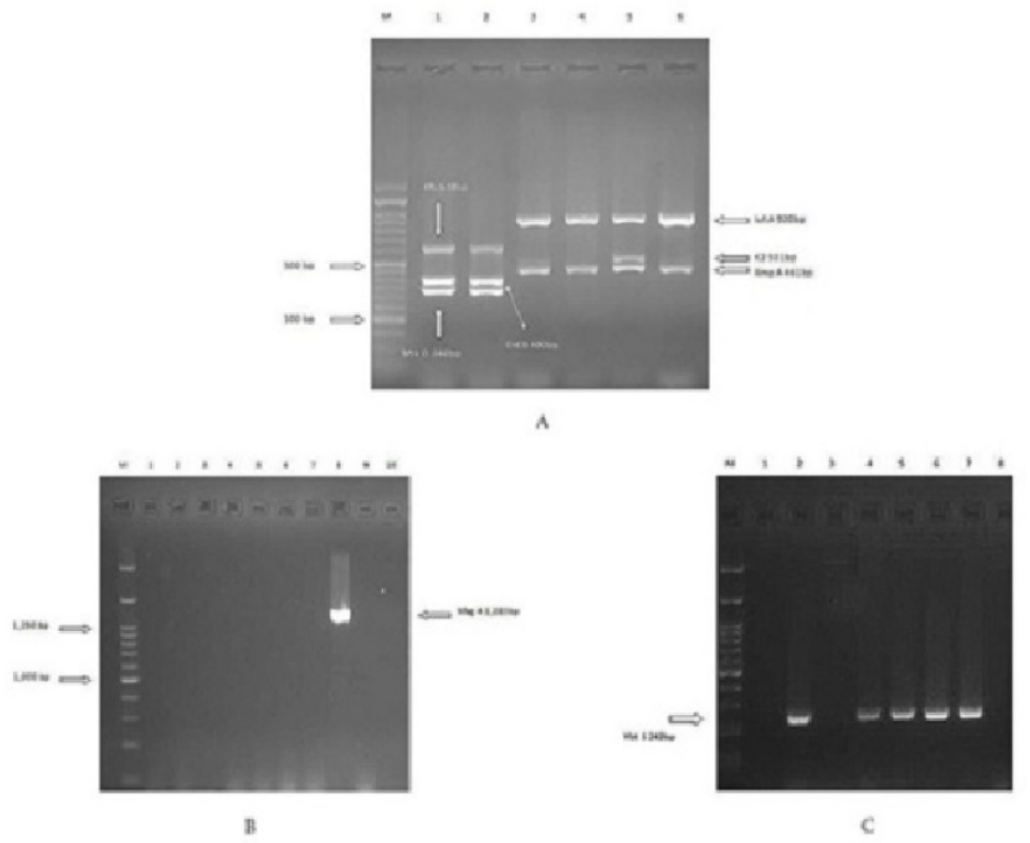

Figure 1. Gels electrophoresis of the PCR products in the *K. pneumoniae* isolates from general hospitals of Kurdistan Province, Iran. A) 2 multiplex PCRs for rmpA, iutA, K2, entB, mrkD and kfu genes. Lane 1: marker 100 bp; Lanes 1, 2: clinical isolates with entB, mrkDand kfu genes; Lanes 3, 4, 6: clinical isolates with iutA and K2 genes; Lane 5: clinical isolate with rmpA, iutA and K2 genes; **B**) PCRs for magA gene. Lane M: marker 1kb; Lane 1: negative control; Lanes 2, 3, 4, 5, 6, 7, 9, 10: clinical isolates ybtS negative;Lane 8: clinical isolate positive ybtS gene; **C**) PCRs for ybtSgene. Lane M: marker 100 bp; Line 1: negative control; Lanes 2, 4, 5, 6, and 7: clinical isolates ybtS positive; Lanes 3, 8: clinical isolates ybtS negative gene.

al. in Brazil reported that all *K. pneumoniae* isolates were ESBL-positive. The prevalence of ESBL-producing clinical isolates is related to different risk factors such as current antibiotic use, resent hospitalization [21].

K. pneumoniae has many virulence factors such as capsular polysaccharide, adhesions and siderophores which contribute to the pathogenicity of these bacteria. Presence of virulence factors in *K. pneumoniae* is important because they are the most prominent cause of death in patients before starting antibiotic therapy [15]. *YbtS, entB, mrkD, magA, kfu, iutA, rmpA and K2* genes are among genes that code virulence factors [22]. Our study focused on detection of *ybtS, entB, mrkD, magA, kfu, iutA, rmpA*, and *K2* genes in ESBL and non-ESBL producing *K. pneumoniae* isolates. The important point in this study is that it was the first study to report the presence of virulencegenes in *K. pneumoniae* isolates in Kurdistan Province, Iran. So far, there has been no report of virulencegenes in *K. pneumoniae* on Google Scholar and PubMed. In the present study, *entB* was determined in 81.43% of the isolates whereas no isolates carried *K2* among all the *K. pneumoniae* isolates taken from the clinical specimens. Nevertheless, *entB* and *K2* werethe highest and lowest prevalent virulence factors in the current study. In this investigation, among the 70

isolates collected from clinical specimens such as blood, tracheal, wound, and urine, 10 isolates (14.3%) were hv-KP isolates.

Frequency of *rmpA* was (n=4, 5.7 %) that all the isolates were ESBL. According to table 2, this is while all the hvKP-isolates had the *entB* gene and ESBL phenotype. In contrast, in previous studies, such as Yu *et al.* in Taiwan, the prevalence of hv-KP, *rmpA,* and *magA* was reported to be 38%, 48% and 17%, respectively; the result of their study showed that strains carrying *rmpA* were significantly associated with hv-Kp [23]. On the other hand, Nahavandinejad *et al.* in northern Iran demonstrated that the hv-KP isolates were not restricted to *magA* [24]. *MagA* was only found in one ESBL isolate that contained the *ybtS, entB, mrkD,* and *kfu* genes. In contrast, *MagA* was much higher than the *magA and K2* genes detected in Korea [25] and Taiwan [8]. These difference between the prevalence of *MagA and K2* could be related to sample type of infection [26]. In the majority of those studies, *K. pneumoniae* was isolated from liver and meninges curtains infections whereas in our study, the isolates were collected from the clinical specimens [2,8]. In a study conducted by Feizabadi *et al.* in Iran on 89 isolates of *K. pneumoniae*, 10 (11.2%) isolates belonged to K1 and 13 (14.6%) isolates belonged to K2 serotypes, respectively [27]. Amraie *et al.* in Shahrekord, Iran, reported low frequency of *MagA* among clinical isolates, which is similar to our results [26]. Prior studies suggested that the magA gene can be infrequently seen in *K. pneumoniae* isolated clinical samples except liver abscesses [26,28]. Compain *et al.* in France designed a multiplex PCR for identifying seven virulence factors and *K1/K2* capsular serotypes of *K. pneumoniae*. The multiplex PCR was used on 65 *K. pneumoniae* isolates between 2004 and 2014, which included 45 clinical isolates identified as hvKP; most isolates (64 /65) were found to possess *mrkD* [14] which is dissimilar to our results. Unfortunately, there has been no report of virulence genes in ESBL and non ESBL *K. pneumoniae*.

As a result of these investigations, the presence of virulence genes in ESBL-producing isolates more than clinical isolates of *K. pneumoniae* lacking ESBL. Our results indicated that there were no statistically significant differences between the ESBL productions and presence of the *ybtS, entB, mrkD, magA, kfu, iutA, rmpA,* and *K2* genes. Moreover, the presence of these genes and variables such as presence of sex, clinical specimen type and hv-KP phenotype between ESBL and non –ESBL *K. pneumoniae* isolates (0.05< p).

5. Conclusions

In conclusion, frequency of ESBL-producing *K. pneumoniae* is increasing now. Detection of virulence factors that positively impact the pathogenicity of *K. pneumoniae* is of immense importance. The results of the current study showed that *entB* was the major virulence factor for *K. pneumoniae* (ESBL and non-ESBL) isolated from the clinical specimens in the hospitals of Kurdistan Province, Iran.

6. Open Access

7. List of abbreviations

CDDT: Combination disk diffusion test; **ESBL**: Extended-spectrum betalactamase; **LPS**: Lipopolysaccharide; **MDR**: Multi-drug resistant; **PCR**: Polymerase Chain Reaction; **UTI**: Urinary tract infection

8. Ethics approval and consent to participate

The study was approved by Kurdistan University of medical science, Iran. All the members were fully informed of the purpose of the investigation, and were informed.

9. Competing interests

The authors declare no conflict of interest.

10. Funding

None

11. Authors' contributions

PS: Study design, doing experiments, data collection, writing; MKT: Study design, writing, critical review; RR: Supervision, study design, writing, critical review; AA: Data collection, data analysis, critical review; SR: Doing experiments, data collection, data analysis, critical review.

12. Acknowledgments

This work was retrieved from the thesis of PhD student, Pegah Shakib and, Kurdistan University of Medical Sciences supported this study.

References

1. Podschun R, Ullmann U.
Klebsiella spp. as nosocomial pathogens: epidemiology, taxonomy, typing methods, and pathogenicity factors.
Clinical Microbiology Reviews. 1998;11:589–603.
2. Fang CT, Chuang YP, Shun CT, Chang SC, Wang JT.
A novel virulence gene in Klebsiella pneumoniae strains causing primary liver abscess and septic metastatic complications.
The Journal of Experimental Medicine. 2004;199:697–705.
Available from: DOI:10.1084/jem.20030857.
3. Azadpour M, Nowroozi J, Goudarzi GR, Mahmoudvand H.
Presence of qacEΔ1 and cepA genes and susceptibility to a hospital biocide in clinical isolates of Klebsiella pneumoniae in Iran.
Trop Biomed . 2015;32(1):109–115.
4. Chander A, Shrestha CD.
Prevalence of extended spectrum beta lactamase producing Escherichia coli and Klebsiella pneumoniae urinary isolates in a tertiary care hospital in Kathmandu, Nepal.
BMC Research Notes. 2013;6:487.
Available from: Doi:10.1186/1756-0500-6-487.
5. Drawz SM, Papp-Wallace KM, Bonomo RA.
New β-lactamase inhibitors: a therapeutic renaissance in an MDR world.
Antimicrobial Agents and Chemotherapy. 2014;58:1835–46.
Available from: Doi:10.1128/aac.00826-13.
6. Kil KS, Darouiche RO, Hull RA, Mansouri MD, Musher DM.
Identification of a Klebsiella pneumoniae strain associated with nosocomial urinary tract infection.
Journal of Clinical Microbiology. 1997;35:2370–4.
7. Lery LM, Frangeul L, Tomas A, Passet V, Almeida AS, Bialek-Davenet S.
Comparative analysis of Klebsiella pneumoniae genomes identifies a phospholipase D family protein as a novel virulence factor.
BMC Biology. 2014;12:41.

Available from: Doi:10.1186/1741-7007-12-41.
8. Yeh KM, Lin JC, Yin FY, Fung CP, Hung HC, Siu LK.
Revisiting the importance of virulence determinant magA and its surrounding genes in Klebsiella pneumoniae causing pyogenic liver abscesses: exact role in serotype K1 capsule formation.
The Journal of Infectious Diseases. 2010;201:1259–67.
Available from: Doi:10.1086/606010.
9. Lin YC, Lu MC, Tang HL, Liu HC, Chen CH, Liu KS.
Assessment of hypermucoviscosity as a virulence factor for experimental Klebsiella pneumoniae infections: comparative virulence analysis with hypermucoviscosity-negative strain.
BMC Microbiology. 2011;11:50.
Available from: Doi:10.1186/1471-2180-11-50.
10. Lin JC, Koh TH, Lee N, Fung CP, Chang FY, Tsai YK.
Genotypes and virulence in serotype K2 Klebsiella pneumoniae from liver abscess and non-infectious carriers in Hong Kong, Singapore and Taiwan.
Gut Pathogens. 2014;6:21.
Available from: Doi:10.1186/1757-4749-6-21.
11. Osman KM, Hassan HM, Orabi A, Abdelhafez AS.
Phenotypic, antimicrobial susceptibility profile and virulence factors of Klebsiella pneumoniae isolated from buffalo and cow mastitic milk.
Pathogens and Global Health. 2014;108:191–9.
Available from: Doi:10.1179/2047773214y.0000000141.
12. Yeh KM, Kurup A, Siu LK, Koh YL, Fung CP, Lin JC.
Capsular serotype K1 or K2, rather than magA and rmpA, is a major virulence determinant for Klebsiella pneumoniae liver abscess in Singapore and Taiwan.
Journal of Clinical Microbiology. 2007;45:466–71.
Available from: Doi:10.1128/jcm.01150-06.
13. Enani MA, El-Khizzi NA.
Community acquired Klebsiella pneumoniae, K1 serotype. Invasive liver abscess with bacteremia and endophthalmitis.
Saudi Medical Journal. 2012;33:782–6.
14. Compain F, Babosan A, Brisse S, Genel N, Audo J, Ailloud F.
Multiplex PCR for detection of seven virulence factors and K1/K2 capsular serotypes of Klebsiella pneumoniae.
Journal of Clinical Microbiology. 2014;52:4377–80.
Available from: Doi:10.1128/jcm.02316-14.
15. Lawlor MS, O'connor C, Miller VL.
Yersiniabactin is a virulence factor for Klebsiella pneumoniae during pulmonary infection.
Infection and Immunity. 2007;75:1463–72.
Available from: Doi:10.1128/iai.00372-06.
16. Beyene G, Tsegaye W.
Bacterial uropathogens in urinary tract infection and antibiotic susceptibility pattern in jimma university specialized hospital, southwest ethiopia.
Ethiopian Journal of Health Sciences. 2011;21:141–6.
Available from: DOI:10.4314/ejhs.v21i2.69055.
17. Ongut G, Daloglu AE, Baysan BO, Daglar D, Ogunc D, Sekercioglu AO.
Evaluation of a chromogenic medium for detection of extended-spectrum-beta-lactamase-producing Escherichia coli and Klebsiella pneumoniae strains.
Clinical Laboratory. 2014;60:1213–5.
18. Cassu-Corsi D, Martins WM, Scheffer MC, Cayô R, Gales AC.
Misidentification of pan drug-resistant Klebsiella pneumoniae clinical isolates as a metallo-β-lactamase producers by the EDTA/DDST test.
The Brazilian Journal of Infectious Diseases. 2015;19:102–4.
Available from: DOI:10.1016/j.bjid.2014.08.008.
19. Ruiz-Garbajosa P, Curiao T, Tato M, Gijón D, Pintado V, Valverde A.
Multiclonal dispersal of KPC genes following the emergence of non-ST258 KPC-producing Klebsiella pneumoniae clones in Madrid, Spain.

The Journal of Antimicrobial Chemotherapy. 2013;68:2487–92.
Available from: DOI:10.1093/jac/dkt237.

20. Ghasemi Y, Archin T, Kargar M, Mohkam M.
A simple multiplex PCR for assessing prevalence of extended-spectrum β-lactamases producing Klebsiella pneumoniae in Intensive Care Units of a referral hospital in Shiraz, Iran.
Asian Pacific Journal of Tropical Medicine. 2013;6:703–8.
Available from: Doi:10.1016/s1995-7645(13)60122-4.
21. Jaskulski MR, Medeiros BC, Borges JV, Zalewsky R, Fonseca ME, Marinowic DR.
Assessment of extended-spectrum β-lactamase, KPC carbapenemase and porin resistance mechanisms in clinical samples of Klebsiella pneumoniae and Enterobacter spp.
International Journal of Antimicrobial Agents. 2013;42:76–9.
Available from: DOI:10.1016/j.ijantimicag.2013.03.009.
22. Brisse S, Fevre C, Passet V, Issenhuth-Jeanjean S, Tournebize R, Diancourt L.
Virulent clones of Klebsiella pneumoniae: identification and evolutionary scenario based on genomic and phenotypic characterization.
PLoS One. 2009;4:e4982.
Available from: DOI:10.1371/journal.pone.0004982.
23. Yu WL, Ko WC, Cheng KC, Lee HC, Ke DS, Lee CC.
Association between rmpA and magA genes and clinical syndromes caused by Klebsiella pneumoniae in Taiwan.
Clinical Infectious Diseases. 2006;42:1351–8.
Available from: Doi:10.1086/503420.
24. Nahavandinejad M, Asadpour L.
Mucoviscosity Determination and Detection of magA and rmpA Genes in Clinical Isolates of Klebsiella pneumoniae in Northern Iran.
Crescent Journal of Medical and Biological Sciences. 2017;4:104–7.
25. Chung DR, Lee HR, Lee SS, Kim SW, Chang HH, Jung SI.
Evidence for clonal dissemination of the serotype K1 Klebsiella pneumoniae strain causing invasive liver abscesses in Korea.
Journal of Clinical Microbiology. 2008;46:4061–3.
Available from: Doi:10.1128/jcm.01577-08.
26. Amraie H, Shakib P, Rouhi S, Bakhshandeh N, Zamanzad B.
Prevalence assessment of magA gene and antimicrobial susceptibility of Klebsiella pneumoniae isolated from clinical specimens in Shahrekord, Iran.
Iranian Journal of Microbiology. 2014;6:311–6.
27. Feizabadi MM, Raji N, Delfani S.
Identification of Klebsiella pneumoniae K1 and K2 capsular types by PCR and quellung test.
Jundishapur Journal of Microbiology. 2013;6:e7585.
Available from: DOI:10.5812/jjm.7585.
28. Fang FC, Sandler N, Libby SJ.
Liver abscess caused by magA+ Klebsiella pneumoniae in North America.
Journal of Clinical Microbiology. 2005;43:991–2.
Available from: Doi:10.1128/jcm.43.2.991-992.2005.