

Clinical characteristics and risk factors for mortality in *Morganella morganii* bacteremia

Ing-Kit Lee¹, Jien-Wei Liu^{1,2}

¹Division of Infectious Diseases, Department of Internal Medicine,
Chang Gung Memorial Hospital-Kaohsiung Medical Center, Kaohsiung Hsien;
and ²Faculty of Medicine, Chang Gung University Medical College, Taiwan

Received: February 22, 2005 Revised: April 1, 2005 Accepted: July 22, 2005

Background and Purpose: To clarify the clinical characteristics and risk factors for mortality of patients with *Morganella morganii* bacteremia.

Methods: Retrospective analyses were undertaken of patients with *M. morganii* bacteremia treated at Chang Gung Memorial Hospital-Kaohsiung, between 2002 and 2003.

Results: Seventy three patients (39 male, 34 female; mean age, 64.43 ± 16.58 years) were included for analyses. At least 1 underlying disease was found in 91.7% of patients. Solid tumors (34.2%) was most frequently encountered. The leading portals of entry of *M. morganii* bacteremia were the urinary tract (37%) and hepatobiliary tract (22%). Of all included cases, 69.9% were community-acquired and 45.2% were of polymicrobial bacteremia. Urinary tract (47.5%) and hepatobiliary tract (30.3%) were the major portals of entry among patients with monomicrobial and polymicrobial *M. morganii* bacteremia, respectively. The overall mortality rate was 38.3%. Susceptibility testing of *M. morganii* isolates showed universal resistance to cephalothin, and high resistance rates to cefuroxime (90.5%) and amoxicillin-clavulanate (95.9%). In contrast to 95.8% of the *M. morganii* isolates being ceftazidime-susceptible, 19.4% were imipenem-resistant. Univariate analyses showed that fatal cases had significantly higher rates of diabetes mellitus (50% vs 20%, $p=0.010$), polymicrobial bacteremia (64.2% vs 33.3%, $p=0.015$) and inappropriate antibiotic treatment (67.8% vs 26.6%, $p=0.001$). Multivariate analysis indicated that inappropriate antibiotic treatment (odds ratio, 4.8, $p=0.002$) was the only independent risk factor for mortality.

Conclusions: *M. morganii* bacteremia frequently occurred secondary to urinary tract or hepatobiliary tract infection, and was associated with a high mortality rate, especially for those not receiving appropriate antibiotic therapy.

Key words: Bacteremia, bacterial drug resistance, microbial sensitivity tests, *Morganella morganii*, mortality

Introduction

Morganella morganii is the only species in the genus *Morganella* which belongs to the tribe *Proteeae* of the *Enterobacteriaceae* family; the other 2 genera in the tribe *Proteeae* are *Proteus* and *Providencia* [1]. Organisms in the tribe *Proteeae* are normally found in soil, water and sewage, and also are part of the normal fecal flora in humans. Among these microorganisms,

Proteus species are the most common human pathogens and cause a variety of community- and hospital-acquired infectious diseases, including urinary tract infection, septicemia and wound infection [2-4]. In contrast, bacteremia due to *M. morganii* remains relatively uncommon. *M. morganii* bacteremia has been reported to occur sporadically either as isolated cases or in series as hospital outbreaks [5-15]. The incidence of *M. morganii* bacteremia, and the clinical features, primary infection sites, underlying diseases and risk factors for mortality of patients with *M. morganii* bacteremia have not been extensively studied. We performed a retrospective study of patients with *M. morganii* bacteremia treated at our hospital.

Corresponding author: Dr. Jien-Wei Liu, Division of Infectious Diseases, Department of Internal Medicine, Chang Gung Memorial Hospital-Kaohsiung Medical Center, 123, Ta-Pei Road, Niao-Sung Hsiang, Kaohsiung Hsien 833, Taiwan.
E-mail: 88b0@adm.cgmh.org.tw

Methods

Patients with *M. morganii* bacteremia treated at Chang Gung Memorial Hospital, Kaohsiung, a 2500-bed tertiary referral medical center in southern Taiwan, between January 1, 2002 and December 31, 2003 were identified by searching the blood culture log book of the Clinical Microbiology Laboratory. The medical charts of these patients were reviewed to collect information regarding demographic characteristics, underlying diseases, primary infection foci of *M. morganii* bacteremia, other concurrent isolated microorganisms, antibiograms, antibiotic therapy, treatment ward and unit, and clinical outcomes.

M. morganii isolates were identified based on the following findings: microscopic recognition of Gram-negative bacilli that fermented mannose but not lactose, maltose and inositol; the presence of indole, tryptophan deaminase, urease, and ornithine decarboxylase; as well as the production of hydrogen sulfide [1,16]. In vitro antimicrobial susceptibility testing was performed on a routine clinical service basis using the Kirby-Bauer disk-diffusion method. The procedures and breakpoint diameters for interpretation were in accordance with National Committee for Clinical Laboratory Standards (NCCLS) [17]. Tested antibiotics included amoxicillin-clavulanate (30 µg per disk), piperacillin (100), cephalothin (30), cefuroxime (30), ceftazidime (30), imipenem (10), amikacin (30), gentamicin (10), ciprofloxacin (5) and trimethoprim-sulfamethoxazole (1.25/23.75). Both intermediate and resistance in susceptibility testing were grouped as resistance. The minimal inhibitory concentrations (MICs) were measured by the Vitek System (BioMérieux Vitek, Hazelwood, MO, USA).

Steroid use was classified based on a documented history of using steroids or if a history of using herbal drugs of unknown ingredients was noted with clinical evidence of Cushingoid habitus (e.g., moon face, buffalo hump, central obesity and paper-thin skin). Polymicrobial bacteremia was defined as isolation of one or more additional microorganisms other than *M. morganii* from blood culture. Bacteremia was considered hospital-acquired if blood culture performed ≥ 72 h after admission was positive for *M. morganii* in a patient who was asymptomatic for bacteremia at admission, or if the *M. morganii* bacteremia had been acquired during stay at another hospital or long-term care facility before admission. Portal of entry of *M. morganii* was identified based on presentations clinically suggestive of the specific inflammatory site.

Inappropriate antibiotic therapy was defined as the use of antibiotics within 3 days of the onset of bacteremia to which all of the isolated microorganisms from blood were non-susceptible in vitro. A recurrent episode of *M. morganii* bacteremia was defined as a repeated growth from blood culture in the same patient during the same hospital stay. In cases of recurrent *M. morganii* bacteremia, only the first episode of bacteremia was included in the analyses. Mortality was defined as death within 14 days after the onset of *M. morganii* bacteremia.

Analysis of differences between fatal and non-fatal groups and between monomicrobial and polymicrobial *M. morganii* bacteremic groups was performed using Student's *t* test or Mann-Whitney *U* test for continuous variables, and chi-squared or Fisher's exact test for dichromatic variables. To determine the independent risk factors for mortality, variables which were significant in the univariate analyses of the fatal and non-fatal *M. morganii* bacteremic groups were entered into a multivariate logistic regression model. A 2-tailed *p* value < 0.05 was considered statistically significant.

Results

During the 2-year study period, 10,639 blood cultures positive for bacterial and fungal growth were recorded, of which 74 (0.69%) were positive for *M. morganii*, 163 (1.53%) positive for *Proteus* spp. and 12 (0.11%) positive for *Providencia* spp. Except for 1 patient who developed perihepatic abscess after choledochocystectomy and had recurrent secondary *M. morganii* bacteremia (only the first episode was counted), all of the patients had a single episode of *M. morganii* bacteremia. Of the 73 *M. morganii* bacteremic patients, 68.4% stayed in a general ward (medical ward, 49.3%; surgical ward, 19.1%), 13.8% in an intensive care unit and 17.8% in emergency services. The demographic and clinical data of the patients are summarized in Table 1. There were 39 males and 34 females. The mean age was 64.43 ± 16.58 years (median, 67 years; range, 1-90 years), and 30 (41.0%) patients were older than 70 years. Of the 73 patients, 67 (91.7%) had more than 1 underlying disease. The 5 leading underlying diseases were solid tumors (34.2%), diabetes mellitus (31.5%), chronic renal failure (31.5%), hypertension (24.6%), and non-neoplastic hepatobiliary disease (20.5%) [including 11 cases of choledocholithiasis, 3 liver cirrhosis, and 1 clonorchiasis]. Renal and/or bladder cancer was the most common solid tumor, accounting for 12.3% of all cases.

Table 1. Demographic and clinical characteristics of 73 patients with *Morganella morganii* bacteremia

Characteristic	Total cases (n = 73)	Fatal group (n = 28)	Non-fatal group (n = 45)	p ^a
	No. (%)	No. (%)	No. (%)	
Age (years)				0.231
Mean (± standard deviation)	64.43 ± 16.58	67.46 ± 16.11	62.55 ± 16.78	
Median (range)	67.0 (1-90)	70.5 (28-90)	66.0 (1-88)	
≥70 years	30 (41.0)	14 (50)	16 (35.5)	0.235
Gender				1.0
Male	39 (53.4)	15 (53.6)	24 (53.3)	
Female	34 (46.6)	13 (46.4)	21 (46.7)	
Underlying disease ^b				
DM	23 (31.5)	14 (50)	9 (20.0)	0.010
HTN	18 (24.6)	7 (25)	11 (24.4)	1.0
Non-neoplastic hepatobiliary disease	15 (20.5)	4 (14.2)	11 (24.4)	0.379
Urolithiasis	5 (6.8)	1 (3.5)	4 (8.88)	0.643
Chronic renal failure	23 (31.5)	12 (42.8)	11 (24.4)	0.124
Solid tumors	25 (34.2)	13 (46.4)	12 (26.6)	0.127
Renal and/or bladder cancer	9 (12.3)	4 (14.2)	5 (11.1)	0.725
Hepatobiliary and/or GI tract cancer	7 (9.5)	4 (14.2)	3 (6.6)	0.417
Cervical cancer	4 (5.4)	1 (3.5)	3 (6.6)	1.0
Others ^c	5 (6.8)	4 (14.2)	1 (2.2)	-
Steroid use	11 (15.0)	5 (17.8)	6 (13.3)	0.739
Source of bacteremia				
Urinary tract	27 (37)	7 (25.0)	20 (44.4)	0.135
Hepatobiliary tract	16 (22)	5 (17.8)	11 (24.4)	0.573
Soft tissue	11 (15.0)	5 (17.8)	6 (13.3)	0.739
Pleuropulmonary system	3 (4.1)	3 (10.7)	0 (0)	1.0
Others	3 (4.1)	1 ^d (3.5)	2 ^e (4.4)	-
Unknown source	13 (17.8)	7 (25)	6 (13.3)	0.225
Community acquisition	51 (69.9)	17 (60.7)	34 (75.5)	0.200
Polymicrobial bacteremia	33 (45.2)	18 (64.2)	15 (33.3)	0.015
Inappropriate antibiotic therapy	31 (42.4)	19 (67.8)	12 (26.6)	0.001

Abbreviations: DM = diabetes mellitus; HTN = hypertension; GI = gastrointestinal

^aFor univariate analyses of the fatal and non-fatal groups.

^bOne patient might have more than 1 underlying disease.

^cIncluding lung cancer (3 cases), nasopharyngeal carcinoma (1 case) and endometrium cancer (1 case).

^dIntravenous catheter exit infection.

^eIncluding fallopian tube abscess and subdural empyema, each 1 case.

M. morganii bacteremia was community-acquired in 51 cases (69.9%) and hospital-acquired in 22 cases (30.1%). In patients with nosocomial *M. morganii* bacteremia, the median duration of hospital stay before the development of bacteremia was 10 days (range, 6-90 days). The portals of entry of *M. morganii* bacteremia included urinary tract (37%), hepatobiliary tract (22%), soft tissue (15%) and pleuropulmonary system (4.1%). Remarkably, abscess in the central nervous system and the fallopian tube caused by *M. morganii* were each found in 1 patient without any underlying disease.

Among the 27 patients with *M. morganii* bacteremia secondary to urinary tract infection, 7 (25.9%) had

renal and/or bladder cancer, 4 (14.8%) urolithiasis, 10 (37.0%) had an indwelling Foley catheter and 3 (11.1%) had percutaneous nephrostomy catheter placement before the development of bacteremia. Among the 16 patients with *M. morganii* bacteremia secondary to hepatobiliary tract infection, 11 (68.7%) had choledocholithiasis, 3 (18.7%) gastrointestinal and/or hepatobiliary tract cancer, and 2 (12.5%) had an indwelling biliary draining tube before the onset of bacteremia.

Among the 73 patients, 40 (54.8%) had mono-microbial and 33 (45.2%) polymicrobial bacteremia. Twelve had at least 3 microorganisms isolated from blood. The concomitantly isolated bacteria, in order of decreasing frequency, were *Enterococcus* spp. (n = 7),

Proteus spp. (n = 7), *Pseudomonas aeruginosa* (n = 6), *Klebsiella pneumoniae* (n = 6), *Escherichia coli* (n = 5), *Klebsiella oxytoca* (n = 3), *Bacteroides fragilis* (n = 3), *Aeromonas* spp. (n = 3), viridans streptococci (n = 2), *Serratia marcescens* (n = 2), group B *Streptococcus* (n = 2), *Enterobacter cloacae* (n = 2), *Citrobacter freundii* (n = 1), *Clostridium perfringens* (n = 1), *Staphylococcus aureus* (n = 1), *Pseudomonas putida* (n = 1) and *Eikenella corrodens* (n = 1).

The results of antimicrobial susceptibility testing are shown in Table 2. The analysis revealed universal resistance to cephalothin, high rates of resistance to cefuroxime (90.5%) and amoxicillin-clavulanate (95.9%), and high rates of susceptibility to ceftazidime (95.8%). Notably, of the 72 *M. morganii* isolates tested against imipenem, 6 had an (MIC) >16 µg/mL and the other 8 had an MIC of 8 µg/mL, resulting in a 19.4% imipenem resistance rate. None of the patients from whom imipenem-resistant *M. morganii* strains were isolated had previously received treatment with a carbapenem. Of the 73 patients, 31 (42.4%) received inappropriate antibiotic therapy, and 28 (38.3%) died of *M. morganii* bacteremia.

Univariate analysis revealed that diabetes mellitus (50% vs 20%, $p=0.010$), polymicrobial bacteremia (64.2% vs 33.3%, $p=0.015$) and inappropriate antibiotic treatment (67.8% vs 26.6%, $p=0.001$) were significantly associated with mortality (Table 1). Multivariate analysis, however, showed that inappropriate antibiotic treatment (odds ratio, 4.8, with 95% confidence interval 1.75-13.27, $p=0.002$) was the only significant independent risk factor for mortality.

The clinical characteristics of the patients with *M. morganii* bacteremia in the monomicrobial and

Table 2. Susceptibilities of 73 *Morganella morganii* isolates determined by disk diffusion method

Antibiotic	No. of susceptible isolates/no. of tested isolates	Susceptible rate ^a (%)
Amoxicillin-clavulanate	3/73	4.1
Piperacillin	59/71	83.0
Cephalothin	0/73	0
Cefuroxime	7/73	9.5
Ceftazidime	69/72	95.8
Imipenem	58/72	80.5
Amikacin	71/72	98.6
Gentamicin	41/72	56.9
Ciprofloxacin	60/71	84.5
Trimethoprim-sulfamethoxazole	34/73	46.5

^aExclusive of intermediate and resistant isolates.

polymicrobial groups are compared in Table 3. In the monomicrobial group, chronic renal failure and solid tumors (each 35%) were the 2 leading underlying diseases, and urinary tract infection (47.5%) and primary bacteremia (20%) were the 2 major infectious diseases. In the polymicrobial group, non-neoplastic hepatobiliary tract disease (36.4%) was the leading comorbidity, and hepatobiliary tract infection (30.3%) and urinary tract infection (24.2%) were the 2 most commonly encountered infectious diseases. Patients in the polymicrobial group were significantly more likely to have non-neoplastic hepatobiliary disease (36.4% vs 7.5%, $p=0.003$), gastrointestinal and/or hepatobiliary tract cancer (21.2% vs 0%, $p=0.003$), inappropriate antibiotic treatment (57.6% vs 30%, $p=0.031$) and fatal outcome (57.6% vs 22.5%, $p=0.003$) [Table 3].

Discussion

The high rate of community acquisition of *M. morganii* bacteremia in the present series was unexpected because previously published case reports and small series suggested that most *M. morganii* bacteremia was nosocomially acquired [2,5-9,14,15]. Patients with *M. morganii* bacteremia in this study, regardless whether it was community- or hospital-acquired, or monomicrobial or polymicrobial infection, tended to be elderly and to have certain comorbidity. The finding that urinary tract (37%) and hepatobiliary tract (22%) were the 2 major portals of entry implies the need for empirical antibiotic coverage of *M. morganii* in severely-ill septic patients with characteristic infectious symptoms until cultures subsequently indicate otherwise. This may be especially important in elderly or immunocompromised patients as well as those with an underlying urinary or hepatobiliary disorder. Physicians should be aware that inappropriate antibiotic therapy is an independent risk factor for fatality in patients with *M. morganii* bacteremia, and that *M. morganii* isolates are always resistant to amoxicillin-clavulanate and first- or second-generation cephalosporins, which are frequently prescribed for urinary tract or hepatobiliary tract infection.

M. morganii has been reported as a causative agent in pneumonia, empyema, pyomyositis, endophthalmitis, surgical wound infection, neonatal sepsis, and spontaneous bacterial peritonitis [1,2,5-15]. Two unusual infections, namely subdural empyema and fallopian tube abscess, resulting in *M. morganii* bacteremia were found in this series. To our knowledge, subdural empyema

Table 3. Comparison of demographic and clinical characteristics between the monomicrobial and polymicrobial groups of patients with *Morganella morganii* bacteremia

Characteristic	Polymicrobial group (n = 33) No. (%)	Monomicrobial group (n = 40) No. (%)	<i>p</i>
Age (years)			0.782
Mean (\pm standard deviation)	64.24 \pm 16.12	64.67 \pm 17.24	
Median (range)	66.0 (28-90)	67.0 (1-90)	
Gender			1.0
Male/female	18 (54.5)/15 (45.5)	21 (53)/19 (47)	
Underlying disease ^a			
DM	10 (30.3)	13 (32.5)	1.0
HTN	8 (24.2)	10 (25)	1.0
Non-neoplastic hepatobiliary disease	12 (36.4)	3 (7.5)	0.003
Urolithiasis	2 (6)	3 (7.5)	1.0
Chronic renal failure	9 (27.2)	14 (35)	0.614
Solid tumors	11 (33.3)	14 (35)	0.807
Renal and/or bladder cancer	3 (9)	6 (15)	0.499
Hepatobiliary and/or GI tract cancer	7 (21.2)	0	0.003
Others ^b	1 (3.0)	8 (20)	-
Source of bacteremia			
Urinary tract	8 (24.2)	19 (47.5)	0.053
Hepatobiliary tract	10 (30.3)	6 (15)	0.157
Soft tissue	6 (18.2)	5 (12.5)	0.530
Pleuropulmonary system	2 (6)	1 (2.5)	0.586
Others	2 ^c (6)	1 ^d (2.5)	-
Unknown source	5 (15.2)	8 (20)	0.761
Community acquisition	24 (72.7)	27 (67.5)	0.798
Inappropriate antibiotic therapy	19 (57.6)	12 (30)	0.031
Mortality	19 (57.6)	9 (22.5)	0.003

Abbreviations: DM = diabetes mellitus; HTN = hypertension; GI = gastrointestinal

^aOne patient might have more than 1 underlying disease.

^bIncluding cervical cancer (4 cases), lung cancer (3 cases), endometrium cancer (1 case) and nasopharyngeal carcinoma (1 case).

^cIncluding intravenous catheter exit infection and fallopian tube abscess, each 1 case.

^dSubdural empyema.

caused by *M. morganii* has not been previously reported, while fallopian tube abscess due to *M. morganii* has been previously reported only once [13]. Neither of these patients had underlying diseases which would predispose them to the development of to *M. morganii* infection.

Virtually all *M. morganii* isolates are capable of producing a variety of inducible chromosomally-encoded AmpC beta (β)-lactamases that are able to hydrolyze penicillins and cephalosporins, and these AmpC enzymes are refractory to inhibition by clavulanic acid [18-23]. As a result, *M. morganii* is resistant to amoxicillin-clavulanate, and first- as well as second-generation cephalosporins [18-23]. Either a third- or fourth-generation cephalosporin, with or without an aminoglycoside (a high amikacin susceptible rate of 98.6% was found in this series), has been recommended for infections caused by AmpC β -lactamase-containing *M. morganii* [24,25].

In practice, carbapenems are often used for the treatment of patients with sepsis due to *Enterobacter* spp., *S. marcescens*, *C. freundii*, *Providencia* spp. and *M. morganii* because of concern that β -lactams, even in combination with other antimicrobials, may lead to a clinical failure because of inducible resistance [24,25]. Inducible β -lactamases and loss of an outer membrane protein have been associated with carbapenem resistance, at least in *K. pneumoniae* [26]. The unexpectedly high imipenem resistance rate of 19.5% among *M. morganii* isolates in this series suggests that the development of resistance among *M. morganii* isolates or other species of *Enterobacteriaceae* might have been under-recognized. Modakkas and Sanyal reported an extensive antibiotic-use-associated 10-fold increase in imipenem resistance among Gram-negative rods inclusive of *M. morganii* within 1 year at a hospital in Kuwait [27]. The relatively high rate of

resistance of *M. morgani* to imipenem in this series suggests the need for extreme care in the monitoring of patients' condition due to possible need for timely antibiotic adjustment when a broad-spectrum carbapenem is used in critically ill patients before *M. morgani* as a culprit pathogen is excluded. This may be especially important when patients have a secondary infection involving an underlying urinary or hepatobiliary abnormality. The differences in the resistance to imipenem and third-generation cephalosporins of *M. morgani* isolates in this study implies the presence of outer membrane protein mutations for imipenem but not hyperproduction of AmpC enzyme, conferring a unique resistance phenotype [25-29]. Ongoing studies of the patterns and mechanisms of antibiotic resistance in *M. morgani* are warranted to determine which adjustments to antibiotic use strategies are most needed.

This retrospective investigation was limited by the non-availability of some important information (i.e., clinical severity grading). This may have led to bias in the analysis of risk factors associated with mortality in *M. morgani* bacteremia.

In summary, this study revealed that *M. morgani* bacteremia frequently occurred secondary to urinary tract or hepatobiliary tract infection. The majority of *M. morgani* bacteremic patients were elderly, had 1 or more comorbid diseases, and community-acquired infection. Polymicrobial infections occurred in a substantial number of *M. morgani* bacteremic patients. *M. morgani* bacteremia was associated with a high mortality rate, especially for those not receiving appropriate antibiotic therapy.

References

- O'Hara CM, Brenner FW, Miller JM. Classification, identification, and clinical significance of *Proteus*, *Providencia*, and *Morganella*. *Clin Microbiol Rev* 2000;13:534-46.
- Kim BN, Kim NJ, Kim MN, Kim YS, Woo JH, Ryu J. Bacteraemia due to tribe *Proteeae*: a review of 132 cases during a decade (1991-2000). *Scand J Infect Dis* 2003;35:98-103.
- Berger SA. *Proteus* bacteraemia in a general hospital 1972-1982. *J Hosp Infect* 1985;6:293-8.
- Watanakunakorn C, Perni SC. *Proteus mirabilis* bacteremia: a review of 176 cases during 1980-1992. *Scand J Infect Dis* 1994;26:361-7.
- Tucci V, Isenberg HD. Hospital cluster epidemic with *Morganella morgani*. *J Clin Microbiol* 1981;14:563-6.
- Williams EW, Hawkey PM, Penner JL, Senior BW, Barton LJ. Serious nosocomial infection caused by *Morganella morgani* and *Proteus mirabilis* in a cardiac surgery unit. *J Clin Microbiol* 1983;18:5-9.
- McDermott C, Mylotte JM. *Morganella morgani*: epidemiology of bacteremic disease. *Infect Control* 1984;5:131-7.
- Boussemart T, Piet-Duroux S, Manouana M, Azi M, Perez JM, Port-Lis M. *Morganella morgani* and early-onset neonatal infection. *Arch Pediatr* 2004;11:37-9. [In French, English abstract].
- Del Pozo J, Garcia-Silva J, Almagro M, Martinez W, Nicolas R, Fonseca E. Ecthyma gangrenosum-like eruption associated with *Morganella morgani* infection. *Br J Dermatol* 1998;139:520-1.
- Arranz-Caso JA, Cuadrado-Gomez LM, Romanik-Cabrera J, Garcia-Tena J. Pyomyositis caused by *Morganella morgani* in a patient with AIDS. *Clin Infect Dis* 1996;22:372-3.
- Mastroianni A, Coronado O, Chiodo F. *Morganella morgani* meningitis in a patient with AIDS. *J Infect* 1994;29:356-7.
- Isobe H, Motomura K, Kotou K, Sakai H, Satoh M, Nawata H. Spontaneous bacterial empyema and peritonitis caused by *Morganella morgani*. *J Clin Gastroenterol* 1994;18:87-8.
- Pomeranz A, Korzets Z, Eliakim A, Pomeranz M, Uziel Y, Wolach B. Relapsing Henoch-Schonlein purpura associated with a tubo-ovarian abscess due to *Morganella morgani*. *Am J Nephrol* 1997;17:471-3.
- Gebhart-Mueller Y, Mueller P, Nixon B. Unusual case of postoperative infection caused by *Morganella morgani*. *J Foot Ankle Surg* 1998;37:145-7.
- Cunningham ET Jr, Witcher JP, Kim RY. *Morganella morgani* postoperative endophthalmitis. *Br J Ophthalmol* 1997;81:170-1.
- Donnenberg MS. *Enterobacteriaceae*. In: Mandell GL, Bennett JE, Dolin R, eds. *Mandell, Douglas and Bennett's principles and practice of infectious diseases*. 6th ed. New York: Churchill Livingstone; 2005:2567-86.
- National Committee for Clinical Laboratory Standards. Performance standards for antimicrobial susceptibility testing. 9th informational supplement. NCCLS document M100-S9. Wayne, Pa: National Committee for Clinical Laboratory Standards; 1999.
- Poirel L, Guibert M, Girlich D, Naas T, Nordmann P. Cloning, sequence analyses, expression, and distribution of ampC-ampR from *Morganella morgani* clinical isolates. *Antimicrob Agents Chemother* 1999;43:769-76.
- Perilli M, Segatore B, de Massis MR, Riccio ML, Bianchi C, Zollo A, et al. TEM-72, a new extended-spectrum beta-lactamase detected in *Proteus mirabilis* and *Morganella morgani* in Italy. *Antimicrob Agents Chemother* 2000;44:2537-9.

20. Tessier F, Arpin C, Allery A, Quentin C. Molecular characterization of a TEM-21 beta-lactamase in a clinical isolate of *Morganella morganii*. *Antimicrob Agents Chemother* 1998; 42:2125-7.
21. Farmer TH, Reading C. Induction of the beta-lactamases of a strain of *Pseudomonas aeruginosa*, *Morganella morganii* and *Enterobacter cloacae*. *J Antimicrob Chemother* 1987;19: 401-4.
22. Yang YJ, Livermore DM. Chromosomal beta-lactamase expression and resistance to beta-lactam antibiotics in *Proteus vulgaris* and *Morganella morganii*. *Antimicrob Agents Chemother* 1988;32:1385-91.
23. Barnaud G, Arlet G, Verdet C, Gaillot O, Lagrange PH, Philippon A. *Salmonella enteritidis*: AmpC plasmid-mediated inducible beta-lactamase (DHA-1) with an ampR gene from *Morganella morganii*. *Antimicrob Agents Chemother* 1998; 42:2352-8.
24. Sahm DF, Storch G. AmpC beta-lactamases. *Pediatr Infect Dis J* 1998;17:421-2.
25. Boyle RJ, Curtis N, Kelly N, Garland SM, Carapetis JR. Clinical implications of inducible beta-lactamase activity in Gram-negative bacteremia in children. *Pediatr Infect Dis J* 2002;21: 935-9.
26. Bradford PA, Urban C, Mariano N, Projan SJ, Rahal JJ, Bush K. Imipenem resistance in *Klebsiella pneumoniae* is associated with the combination of ACT-1, a plasmid-mediated AmpC beta-lactamase and the loss of an outer membrane protein. *Antimicrob Agents Chemother* 1997;41:563-9.
27. Modakkas EM, Sanyal SC. Imipenem resistance in aerobic gram-negative bacteria. *J Chemother* 1998;10:97-101.
28. Bornet C, Davin-Regli A, Bosi C, Pages JM, Bollet C. Imipenem resistance of *Enterobacter aerogenes* mediated by outer membrane permeability. *J Clin Microbiol* 2000;38: 1048-52.
29. Tzouvelekis LS, Tzelepi E, Kaufmann ME, Mentis AF. Consecutive mutations leading to the emergence in vivo of imipenem resistance in a clinical strain of *Enterobacter aerogenes*. *J Med Microbiol* 1994;40:403-7.