

Nursing home-acquired pneumonia: a 10 year single-centre experience

E Polverino,¹ P Dambrava,¹ C Cillóniz,¹ V Balasso,² M A Marcos,³ C Esquinas,¹ J Mensa,² S Ewig,⁴ A Torres¹

¹Respiratory Department, Hospital Clínic-IDIBAPS, Barcelona, Spain

²Servei de Malalties Infeccioses, Hospital Clínic, Barcelona, Spain

³Servei de Microbiologia, Hospital Clínic, Barcelona, Spain

⁴Thoraxzentrum Ruhrgebiet Kliniken für Pneumologie und Infektiologie Ev. Krankenhaus Herne und Augusta-Kranken-Anstalt, Bochum, Germany

Correspondence to

Professor Antoni Torres, Institut Clínic de Pneumologia i Cirurgia Toràcica, Hospital Clínic, Villarroel 170, E-8036 Barcelona, Spain; atorres@clinic.ub.es

Received 3 August 2009

Accepted 28 January 2010

ABSTRACT

Background Pneumonia among nursing home (NH) residents has increased considerably in recent years, but it remains unclear whether it should be considered as community-acquired pneumonia (CAP) or a new category of infection.

Methods 150 consecutive cases of NH-acquired pneumonia (NHAP) (from 1 February 1997 to 1 July 2007) were analysed.

Results Patients (median age, 82 years; range, 77–87 years) showed numerous co-morbidities, (neurological, 55%; pulmonary, 38%; cardiac, 35%) and severe disability for daily activities (partial, 32%; total, 31%). Cases of NHAP were mainly classified as mild to moderate according to the CRB-65 score (CRB-65 classes 0–1 and 2, 41% each). In-hospital and 30-day mortality were 8.7% and 20%, respectively. Aetiology was defined in 57 cases (38%). The most common isolates were *Streptococcus pneumoniae* (58%), *Enterobacteriaceae* (Gram-negative bacteria (GNB)) (9%), atypical bacteria (7%), respiratory viruses (5%), methicillin-resistant *Staphylococcus aureus* (MRSA) (5%) and *Legionella pneumophila* (5%). The most frequent causes of treatment inadequacy were use of β -lactams alone (25%) and lack of aspiration assessment (15%). Prognostic factors of 1-month mortality were neurological comorbidities (OR 4.5; 95% CI 1.3 to 15.7; $p=0.020$), septic shock (OR 6.6; 95% CI 1.3 to 34.0; $p=0.025$), pleural effusion (OR 3.6; 95% CI 1.1 to 11.7; $p=0.036$) and isolation of GNB or MRSA (OR 16.4; 95% CI 2.1 to 128.9; $p=0.008$).

Conclusions The patients show clinical characteristics (eg, age and co-morbidities) comparable with those with hospital-acquired pneumonia. However, microbiological and mortality data of patients with NHAP are more similar to the data of those with CAP. Isolation of GNB or MRSA was associated with increased mortality risk. CAP empirical antibiotic coverage is still indicated in NHAP, although specific risk factors for multidrug-resistant infections should be assessed on an individual basis.

INTRODUCTION

Nursing home-acquired pneumonia (NHAP) is probably the largest subgroup of healthcare-associated pneumonia (HCAP), and the number of cases has increased in recent decades, with the worldwide diffusion of long-term care facilities (LTCFs).¹ Moreover, the number of older individuals living in nursing homes (NHs) is expected to increase dramatically in the next 30 years, as 40% of adults will probably reside in an LTCF in later life.² Pneumonia is the second most common infection in NH and the leading cause of mortality and

hospitalisation.^{3–7} Much information has been gathered on NHAP since the 1970s^{6–8}: patients with NHAP are usually elderly, with multiple diseases (eg, cardiovascular, respiratory and neurological) and poor functional status.^{9–11} The clinical presentation of NHAP is often unusual,^{6 7 9 12 13} with frequent extrapulmonary manifestations (mental confusion and gastrointestinal disorders),¹⁴ and the clinical presentation may be worse than in community-acquired pneumonia (CAP) (eg, hypoxaemia and altered consciousness).^{11 15 16} The mortality rate of NHAP is close to that of hospital-acquired pneumonia (HAP)^{7 9 11 15–17} (20–40%), while its annual incidence is 30-fold that of the general population and 11-fold that for the elderly (≥ 75 years).^{4 7 18 19}

A major concern has emerged regarding the microbial aetiology of NHAP, since two large retrospective studies on HCAP have reported an elevated incidence of pathogens common in nosocomial infections (Gram negative bacilli (GNB), or methicillin-resistant *Staphylococcus aureus* (MRSA))^{10 20} and, particularly, of multi-drug-resistant (MDR) microorganisms,^{21 22} which may justify nosocomial antibiotic coverage on admission.¹

In contrast, two prospective European studies described *Streptococcus pneumoniae* and *Haemophilus influenzae* as the most frequent pathogens in NHAP.^{9 11 17}

Therefore, the microbial aetiology of NHAP is still under debate, while the effect of comorbidities, functional status, abnormal clinical presentation and microbial aetiology on the high mortality of HCAP is unclear.

The first objective of our study was to investigate microbial aetiology in NHAP and, particularly, the frequency of MDR microorganisms. Hence, the adequacy of empirical antibiotic therapy in our hospital was also investigated in order to review the current antibiotic treatment recommendations.

Secondly, we analysed possible risk factors for MDR infections, such as demographic, clinical and biochemical data, and severity scores.

Lastly, we investigated possible prognostic factors of mortality, including demographic, clinical and biochemical data on admission, and microbial aetiology.

MATERIALS AND METHODS

Study population

We prospectively studied all consecutive cases of NHAP admitted to Hospital Clínic, Barcelona, Spain, from 1 February 1997 to 1 July 2007.

In Spain, NH are currently considered institutions dedicated to assist individuals unable to perform routine daily activities autonomously, including toileting, eating and mobility. NH assistance usually includes part-time medical and physiotherapy support and full-time nursing care.

The diagnosis of pneumonia was made by the Emergency department (ED) doctor and confirmed by the respiratory physician in charge of recruitment and data collection for the study. The choice of empirical antibiotic treatment was taken exclusively by the attending physician. Immunosuppressed patients (neoplasia, severe haematological disorders, HIV, immunosuppressant treatment, chemotherapy in the last year) were excluded.

Data collection

Demographic data, comorbid illness (eg, chronic cardiovascular diseases, diabetes mellitus, chronic liver disease, renal failure, dementia and neurological diseases), previous antibiotic treatment and relevant data from the clinical history (eg, use of inhaled/systemic corticosteroids, influenza and pneumococcal vaccinations, smoking, alcohol intake, previous pneumonia, aspiration evidence and antibiotic allergies) were recorded in an Access database. Neurological disorders include degenerative diseases, multiple sclerosis, amyotrophic lateral sclerosis, Parkinson disease, Down syndrome, stroke and postanoxia brain injury. Autonomy for routine daily activities, including toileting, eating and mobility, was classified as follows: total (no need for external help), partial (need for partial help) and none (totally dependent on external help).

The following parameters were also recorded on admission: days of clinical course before admission, time elapsed in ED, clinical symptoms, vital signs, laboratory data, chest x-ray (number of affected lobes, infiltrate radiographic pattern and localisation, presence of pleural effusion/atelectasis/cavitations), and PSI (Pneumonia Severity Index) and CRB-65 (confusion, respiratory rate, low blood pressure, age ≥ 65 years) prognostic scales.

Relevant data on clinical course were recorded, including treatment (antibiotics and systemic corticosteroids, time of first doses from admission, compliance with the American guidelines for CAP management²³), length of hospital stay, oxygen (arterial oxygen pressure (PaO₂)/fractional inspired oxygen (FiO₂)) and ventilatory support (mechanical ventilation, non-invasive ventilation), day of clinical stability according to American Thoracic Society (ATS)/Infectious Diseases Society of America (IDSA) criteria,²³ pulmonary (empyema, respiratory distress, pleural parapneumonic effusion, pneumothorax, surgical pleural draining) and extrapulmonary complications (cardiac arrhythmias, septic shock, acute renal failure, meningitis, endocarditis, SIADH (syndrome of inappropriate antidiuretic hormone secretion), positive/negative *Clostridium* diarrhoea, antibiotic secondary effects), in-hospital and 1-month mortality rates and cause of death, and treatment failure.²³

We considered the treatment prescribed during the first 24 h of hospitalisation to be the initial treatment. An antibiotic regimen was defined as ATS adherent when the chosen antibiotics followed the recommendations included in the 2007 ATS guidelines, regardless of any additional antibiotic received.²³

Appropriate first-line CAP antibiotic treatments were considered to be β -lactam+macrolide or a quinolone alone. Where aspiration was suspected, amoxicillin-clavulanic acid or piperacillin-tazobactam was considered appropriate antibiotic treatment.

Microbiological data

Within the first 24–48 h after admission, regular samples of sputum, blood for two cultures and serum for paired serology

were taken (on admission and 4–6 weeks thereafter) for atypical pathogens (*Chlamydia pneumoniae*, *Mycoplasma pneumoniae*, *Coxiella burnetii*) and respiratory viruses. Tests for detection of urinary antigens for *Legionella pneumophila* and *S pneumoniae* were systematically performed.

Respiratory secretion samples, including spontaneous sputum or bronchoalveolar lavage (BAL) fluid, fibreoptic bronchial aspirates (FBAS), tracheobronchial aspirates (TBAS) and pleural fluid, when available, were collected for Gram and Ziehl–Nielsen stains and for cultures for bacterial, fungal and mycobacterial pathogens.

Sample processing

Sample processing techniques are described in previous studies by our group.²⁴

Diagnostic criteria

The aetiology of pneumonia was classified as presumptive if a valid sputum sample yielded ≥ 1 predominant bacterial strains. Aetiology was considered definite if one of the following criteria was met: (1) blood cultures yielding a bacterial or fungal pathogen (in the absence of an apparent extrapulmonary focus); (2) pleural fluid cultures yielding a bacterial pathogen; (3) seroconversion (ie, a fourfold increase in immunoglobulin G (IgG) titres) for *C pneumoniae* and *L pneumophila* $>1:128$, *C burnetii* $>1:80$, and respiratory viruses (ie, influenza viruses A and B, parainfluenza viruses 1–3, respiratory syncytial virus, adenovirus); (4) a positive urinary antigen for *L pneumophila* or *S pneumoniae*; and (5) bacterial growth in cultures of TBAS or FBAS $>10^5$ cfu/ml and in BAL $>10^4$ cfu/ml. A diagnosis of probable aspiration was made in cases of witnessed aspiration or in the presence of risk factors for aspiration (severely altered consciousness, abnormal swallowing).

We considered an uncommon microbial aetiology for CAP when GNB (ie, *Escherichia coli*, *Klebsiella pneumoniae*, etc), *Pseudomonas aeruginosa* or MRSA were isolated.

STATISTICS

Categorical variables were described using counts and percentages. Continuous variables were expressed as the mean \pm SD, or median and IQR for abnormally distributed data (Kolmogorov–Smirnov test). Relationships between categorical variables were studied using the χ^2 test, or Fisher exact test, when necessary. Comparison of continuous variables between two groups was carried out using the t test for unpaired data once normality was demonstrated; otherwise, the non-parametric test (Mann–Whitney U test) was used. Univariate and multivariate logistic regression analyses were conducted to identify variables predictive of patients with potential first-line CAP antibiotic-resistant microorganisms (GNB or MRSA) (dependent variable). The independent variables were age, sex, length of stay, pneumonia in the previous year, suspected aspiration, inhaled corticosteroids, heart failure, chronic renal failure, diabetes mellitus, chronic liver disease, neurological disorders, chronic respiratory diseases, smoking, alcohol, autonomy for daily activities, pneumococcal vaccination, influenza vaccination, systemic corticosteroids, fever, dyspnoea, acute renal failure, shock, PCR, leucocytes $>12 \times 10^9/l$, PaO₂/FiO₂ <200 , cavitation, atelectasis, pleural effusion, more than two affected lobes, PSI score and CRB-65 classes. Univariate and multivariate logistic regression analyses were performed to predict 30-day mortality (dependent variable). Independent variables were as above, including possible first-line CAP antibiotic-resistant

microorganisms (GNB or MRSA). Variables that showed a significance in the univariate analysis ($p < 0.1$) were included in the multivariate logistic regression backward stepwise model to determine which of them were independently related to outcome. The Hosmer–Lemeshow goodness-of-fit test was performed to assess the overall fit of the model. All analyses were performed using SPSS 16 for Windows; a two-tailed p value of < 0.05 was considered statistically significant.

RESULTS

Study population

We analysed 150 consecutive cases of NHAP (median age 82 years (77–87 years); males, 49%); Table 1 shows the main characteristics.

Median length of stay was 8 days (5–13 days). Only 20 patients (13%) were admitted to the intensive care unit (ICU); their median length of stay was 11 days (9–23 days). Tables 2 and 3 show data on clinical presentation on admission (symptoms, analytical data and radiographic patterns) and severity indexes, respectively. NHAP were mainly classified as mild to moderate according to the CRB-65 score (CRB-65 classes 0–1 and 2, 41% each) but as moderate to severe by the Fine score (Fine classes 4 and 5, 33% and 53%) due to the weight of age in the PSI calculation.

In-hospital mortality was 8.7% ($n=13$) globally and 20% ($n=30$) after 1 month. Deceased patients had been mainly hospitalised in a ward ($n=23$; 77%); only five (17%) were in an ICU. The most frequent respiratory complication was empyema ($n=5$), followed by acute respiratory distress syndrome ($n=2$) and pleural effusion ($n=2$). Extrapulmonary complications included renal failure in nine cases (9%), cardiac arrhythmia in six (6%) and septic shock in five (5%). Clinical stability was reached after a median of 6 days (4–9 days) of hospitalisation.

Aetiology was defined in 38% of cases ($n=57$) (table 4). The most common isolates were *S pneumoniae* (58%) and

Table 1 Main characteristics of the study population, n (%)*

Smoking habit	
Non-smokers	83 (66)
Smokers	12 (10)
Ex-smokers	31 (25)
Vaccinations	
Pneumococcal vaccination	15 (15)
Influenza vaccination	75 (75)
Autonomy for daily activities	
Full	36 (31)
Partial	38 (32)
Previous pneumonia in the last year	23 (18)
Inhaled corticosteroids	25 (19)
Systemic corticosteroids	4 (3)
Co-morbidities	
Heart failure	47 (35)
Chronic renal failure	10 (8)
Diabetes mellitus	28 (21)
Chronic liver disease	3 (2)
Neurological disorders	73 (55)
Chronic respiratory diseases	51 (38)

Chronic respiratory diseases include: asthma (4%), chronic bronchitis (9%), chronic obstructive pulmonary disease (14%), ex tuberculosis sequelae (4%), pulmonary hypertension (1%) and others (4%).

Neurological disorders include: degenerative diseases, multiple sclerosis, amyotrophic lateral sclerosis, Parkinson disease, Down syndrome, vascular cerebral accidents, postanoxia brain injury and dementia.

*Percentages of all cases with available information.

Table 2 Clinical presentation and laboratory data on admission

Symptoms, n (%)*	
Previous 'common cold' symptoms	49 (38)
Fever	106 (79)
Chills	46 (36)
Cough	107 (80)
Purulent phlegm	66 (50)
Pleuritic pain	33 (25)
Dyspnoea	102 (76)
Nausea/vomiting	12 (12)
Altered mental status	67 (50)
Acute renal failure	22 (17)
Shock	14 (10)
Radiographic features	
Cavitation	1 (1)
Atelectasis	8 (6)
Pleural effusion	27 (20)
Alveolar infiltrate pattern	119 (77)
Interstitial infiltrate pattern	1 (1)
Mixed infiltrate pattern	3 (2)
Number of affected lobes, 1	83 (61)
Number of affected lobes, ≥ 2	51 (38)
Vital signs, mean \pm SD	
Respiratory rate	29 \pm 7
Heart rate	96 \pm 19
Systolic blood pressure, mm Hg	129 \pm 28
Diastolic blood pressure, mm Hg	69 \pm 14
Analytical data	
Leucocytes $> 12 \times 10^9/l$, n (%)	76 (56)
Creatinine, mg/dl	1.5 \pm 0.2
C-reactive protein, mg/dl	20 \pm 13
Platelets, $\times 10^3/l$	277 \pm 111
Na, mEq/l	138 \pm 7
K, mEq/l	4.2 \pm 0.7
Haematocrit, %	38 \pm 6
Serum proteins, g/l	60 \pm 6
Albumin, g/l	33 \pm 7

*Percentages of all cases with available information.

Enterobacteriaceae (*E coli* and *K pneumoniae*) (9%). *L pneumophila*, respiratory virus and MRSA were isolated in three cases each (5%). Mixed aetiology was detected in four patients, including two cases of *S pneumoniae* and a respiratory virus, one of *E coli* and *C pneumoniae*, and one of MRSA and *K pneumoniae*. *H influenzae* was isolated in only two patients (4%).

An antibiotic resistance pattern was available in 32 patients, including 22 cases of *S pneumoniae*, 3 MRSA, 1 *P aeruginosa*, 2 *E coli*, 1 *K pneumoniae*, 2 *H influenzae* and 1 *Providencia stuartii*.

Microbial aetiology in deceased patients was determined in only 10 cases (33%) and did not differ significantly from that of survivors, since *S pneumoniae* was the most frequently isolated microorganism ($n=5$; 50%) followed by GNB (2 cases of *K pneumoniae*, 1 *E coli* and 1 *P stuartii*) and MRSA ($n=1$).

Initial antibiotic treatment was aimed at CAP coverage, except for six patients who received a broad-spectrum antibiotic (ie, carbapenem), and included combined β -lactam+quinolone (30%), β -lactam alone (25%), quinolone alone (17%) and combined β -lactam+macrolide (11%). Accordingly, the most frequent causes of empiric antibiotic inadequacy were the use of β -lactams alone and lack of aspiration assessment in 25% and 15% of cases, respectively.

Table 3 Severity indices

	n (%)*
Acute respiratory failure	
PaO ₂ /FiO ₂ <200	16 (16)
Basal SaO ₂ <92%	61 (54)
Acute renal failure (creatinine >1.5 mg/dl)	28 (21)
Shock	14 (10)
Multilobar infiltration	51 (38)
2 lobes affected	42 (31)
≥3 lobes affected	9 (7)
Admission to ICU	13 (9)
Admission to intermediate care unit	7 (5)
Fine score, 1–2	9 (6)
Fine score, 3	12 (8)
Fine score, 4–5	129 (86)
CRB-65 score, 0–1	61 (41)
CRB-65 score, 2	61 (41)
CRB-65 score, 3–4	28 (19)
Pneumonia severity index (PSI), mean±SD	137±28

*Percentages of all cases with available information. CRB-65, pneumonia severity score including: consciousness, respiratory rate, blood pressure, 65 years of age cut-off; ICU, intensive care unit.

With regard to antibiotic resistance, we observed that initial antibiotic therapy was inappropriate in 12 patients (38%); the antibiotic was changed on admission but there were no significant changes in mortality (only three patients with initial inadequate antibiotic therapy died during hospitalisation).

Microbial aetiology

Demographic, clinical and biochemical variables were compared for patients with typical CAP microbiology (ie, *S pneumoniae*, *H influenzae*, etc) (n=47) and those with potential first-line CAP antibiotic-resistant microorganisms (GNB or MRSA) (n=11). No statistically significant differences were found between the two groups except for PSI score (132.6±25.3 for patients with typical CAP vs 157.7±30.0 for patients with GNB or MRSA; t test; p=0.014) and CRB-65, which was higher in patients with GNB or MRSA (classes 3–5: 13% for patients with typical CAP vs 54% for patients with GNB or MRSA; χ^2 ; p=0.007). Statistically significant variables in the univariate analysis were PSI scoring (+1 point increase; OR 1.04; 95% CI 1.01 to 1.07; p=0.024),

CRB-65 classes 3–5 (OR 12.5; 95% CI 2.0 to 78.0; p=0.007) and length of stay (+1 day increase; OR 1.08; 95% CI 0.99 to 1.17; p=0.075). No independent predictors of GNB or MRSA were found in the multivariate analysis.

One-month mortality

Demographic, clinical and biochemical variables were compared between patients who were deceased at 1 month (n=30) and survivors. No significant differences were found between the two groups except for neurological co-morbidities (76% for patients who had died at 1 month vs 49% for survivors; χ^2 ; p=0.023), and autonomy for daily activities (full: 6% for patients who died at 1 month vs 37% for survivors; χ^2 ; p=0.023). In the multivariate analysis (table 5), the independent predictors of 1-month mortality were neurological diseases (OR 4.5; 95% CI 1.3 to 15.7; p=0.020), septic shock (OR 6.6; 95% CI 1.3 to 34.0; p=0.025), pleural effusion (OR 3.6; 95% CI 1.1 to 11.7; p=0.036) and isolation of GNB or MRSA (OR 16.4; 95% CI 2.1 to 128.9; p=0.008) (table 5).

The χ^2 goodness-of-fit analysis demonstrated the model's adequacy (p>0.05).

DISCUSSION

The peculiarities of NH populations and the increased risk of mortality have led NHAP to be considered a separate clinical entity, thereby justifying specific recommendations for clinical management^{25–27} and, recently, inclusion among HCAP.¹ As in the literature, our population showed advanced age, numerous comorbidities (particularly neurological disorders), poor autonomy for daily activities (up to 70% of patients were partially or totally dependent) and, frequently, atypical clinical presentation (extrapulmonary manifestations). The role of age, comorbidities and functional status in this population thus appears to have a considerable effect on pneumonia severity and mortality.²⁸ Many authors have shown that patients' functional status before admission is one of the most important prognostic factors for mortality^{6, 29–32} and may considerably influence the decision of site of care (NH vs hospital, ICU admission, etc) and aspects of clinical management (diagnostic procedures and life-prolonging treatments).^{7, 26, 33, 34} We also observed that a history of neurological disorders (a major cause of the inability to perform daily activities) was an important prognostic factor of mortality. The highest mortality rate was recorded among

Table 4 Bacteriological findings of 57 nursing home patients (38% of total population) with either probable or definitive aetiology of a pneumonia episode

	n (%)	Sputum	Blood	TBAS, BAL	Pleural fluid	Serology	Urinary antigen	N-P swab
<i>S pneumoniae</i>	33 (22)	9	6	3	4		19	
<i>H influenzae</i>	2 (1)	2	0	1	0			
MRSA	3 (2)	2	1	1	0			
<i>P aeruginosa</i>	2 (1)	2	0	0	0			
<i>K pneumoniae</i>	2 (1)	0	1	2	0			
<i>E coli</i>	3 (2)	0	1	2	1			
<i>C pneumoniae</i>	1 (1)					2		
<i>M pneumoniae</i>	2 (1)					2		
<i>C burnetii</i>	1 (1)					1		
<i>L pneumophila</i>	3 (2)	0	0	0	0		3	
Virus	3 (2)					3		2
<i>P stuartii</i>	1 (1)	1						
<i>Candida albicans</i>	1 (1)	1	0	1	0			
Total	57 (38)	17	9	10	5	8	22	2

Note: percentages refer to the total number of patients (n=150).

BAL, bronchioalveolar lavage; MRSA, methicillin-resistant *Staphylococcus aureus*; N-P swab, naso-pharyngeal swab; TBAS, tracheobronchial aspirate.

Table 5 Analysis of prognostic factors of 1-month mortality: significant univariate and multivariate associations

	Univariate analysis			Multivariate analysis		
	OR	95% CI	p Value	OR	95% CI	p Value
Autonomy for daily activities			0.078			
Full	1.0	—	—			
Partial	8.7	1.0 to 75.3	0.050			
None	11.5	1.4 to 95.6	0.024			
Neurological diseases	3.3	1.1 to 9.9	0.029	4.5	1.2 to 15.7	0.020
Septic shock	3.0	0.9 to 10.1	0.078	6.6	1.2 to 34.0	0.025
Pleural effusion	2.7	0.9 to 7.9	0.063	3.6	1.1 to 11.7	0.036
Pathogens			0.036			0.028
Typical CAP	1.0	—	—	1.0	—	—
GNB+MRSA	6.5	1.4 to 29.4	0.015	16.4	2.1 to 128.9	0.008
Unknown aetiology	3.0	1.0 to 8.7	0.043	3.0	0.6 to 14.4	0.165
PSI scoring, +1 point	1.02	1.01 to 1.05	0.029			

Typical CAP pathogens include: *S pneumoniae*, *H influenzae*, *L pneumophila*, *M pneumoniae*, *C burnetii*, *C pneumoniae*. GNB include: *P aeruginosa*, *E coli*, *K pneumoniae*, *P stuartii*. Neurological disorders include: degenerative diseases, multiple sclerosis, amyotrophic lateral sclerosis, Parkinson disease, Down syndrome, vascular cerebral accidents, postanoxia brain injury and dementia. CAP, community-acquired pneumonia; GNB, Gram-negative bacilli; MRSA, methicillin-resistant *Staphylococcus aureus*; PSI, pneumonia severity index; '+1 point' indicates a PSI increase of one point.

patients admitted to the ward, suggesting that functional status, premorbid condition and, possibly, ethical considerations limited ICU admission of patients with poor life expectancy.

The mortality rate of our population (8%) was very similar to^{14 35} or lower³⁶ than that reported for CAP in the general population and in the elderly. In contrast, many studies on NHAP^{9 11 28} and the recent North American series on HCAP report higher mortality rates (up to 40%).^{15 16} Interestingly, the Spanish study by Carratalà on HCAP, including 32 NH patients (25.4% of all HCAP), shows a mortality rate very close to that of this study (10.3% for HCAP and 4% for CAP).¹⁷ This similarity suggests two considerations: first, the considerable differences among these studies may denote a geographic variation of pathogens, virulence and antibiotic resistances. Secondly, no unique definition of NH is currently available worldwide and it may not be possible given the organisational differences in local healthcare systems and LTCFs in different countries. Therefore, both the geographic distribution of pathogens and differences in NH populations from different countries may partly explain such remarkable differences in mortality.

A central issue regarding NHAP and HCAP is microbial aetiology and, consequently, the empirical antibiotic treatment to use in clinical practice. Before guidelines for HAP, ventilator-associated pneumonia (VAP) and HCAP, published in 2005,¹ recommendations for NHAP antibiotic treatment closely resembled CAP guidelines.^{23 37} Since the North American studies^{15 16} reported a very high incidence of MDR infections among HCAP, an intense debate arose on the microbial aetiology of NHAP. Subsequently, American guidelines suggested empirically treating HCAP as a nosocomial infection.¹

However, analysing the American retrospective studies on HCAP,^{15 16} it is clear that the high rates of ICU admission, ventilatory support and mortality (pneumonia severity), the high incidence of GNB, even in patients with CAP, and the scarce information on microbiological methods make these data poorly comparable with ours.

The literature shows that, with the exception of two American studies on elderly patients with severe NHAP,^{10 11} no other studies confirm the hypothesis of a nosocomial pattern in NHAP and HCAP.^{9 11 17} Venditti *et al*³⁸ show that receiving empirical antibiotic treatment not recommended by the latest HCAP guidelines was independently associated with increased

mortality. Surprisingly, no microbiological data are shown in that study, suggesting that clinical conditions before hospitalisation, comorbidities or any other possible confounding factor may explain these results.

This is one of the largest NHAP series of the last decade and clearly shows that, in Spain, *S pneumoniae* is still the most frequent organism causing pneumonia. Unfortunately, the rate of positive microbiological findings is fairly low, but similar to many other series of NHAP⁷ for different reasons, such as the following: (1) a poor cough reflex and an altered mental status considerably reduce availability of sputum samples in NH patients; (2) blood cultures are usually performed only in patients with fever, and elderly patients commonly have fewer temperature alterations in response to infection than younger individuals; and (3) pneumococcal and *L pneumophila* urinary antigens are the most common source of aetiological diagnosis but were introduced only in late 2000. Some infrequent bacteria, such as *P aeruginosa* and MRSA, can be easily detected even in poor-quality biological samples (ie, sputum), thereby increasing the number of cases with known aetiology.

It is also worth noting that empirical treatment was mainly concordant with CAP guidelines²³ and, depending on antibiotic resistance, was inappropriate only in a few cases, with no increased mortality. Moreover, the microbial pattern did not differ between survivors and deceased patients, showing that the effect of aetiology on mortality in our series is probably slight.

However, it is important to underline that isolation of unusual microorganisms such as GNB or MRSA (potentially not susceptible to first-line CAP antibiotic treatment), which are more frequent in patients with higher severity scores, was associated with a considerable increase in the mortality risk.

Therefore, we still need to be cautious with therapeutic recommendations: a careful evaluation of possible risk factors for MDR infections in patients with NHAP, such as previous antibiotic treatment, recent hospitalisation or advanced chronic respiratory disease (ie, bronchiectasis or chronic obstructive pulmonary disease) and functional status before admission, appears essential to guide the use of broad-spectrum antibiotics on an individual basis.³¹ Moreover, functional status before admission should always be investigated in the process to decide diagnostic procedures and site of care.

The following are possible limitations of this study:

1. The introduction of the urinary antigen detection method for *S pneumoniae* and for *L pneumophila* only in 2000 may have influenced the rate of aetiological findings in the first 3 years of this series.
2. The initial study design did not consider a case-control match with patients with CAP that might have complemented clinical and microbiological information on NHAP.
3. This is a single-centre Spanish study and our data may not be representative of Europe.

In conclusion, our NH patients appeared more similar to those affected by HAP in terms of age, functional status and comorbidities; however, low mortality was recorded. A 'community' microbial pattern was observed in NHAP, with the only exception of *Enterobacteriaceae* being slightly more frequent than in CAP. Additionally, isolation of GNB and MRSA was associated with a considerable increase in mortality risk.

These findings suggest that 'community-based' empirical antibiotic treatment is still indicated in NHAP, but additional risk factors for MDR infections (eg, previous antibiotic treatment and recent hospitalisation) and the risk of aspiration should always be assessed on an individual basis in order to guide the selection of broad-spectrum antibiotic treatment.

Further investigation with sound microbiological methodology is needed in NHAP to properly evaluate risk factors for MDR infections and optimise empirical antibiotic therapy.

Competing interests None.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1. **American Thoracic Society.** Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. *Am J Respir Crit Care Med* 2005;**171**:388–416.
2. **Richards C.** Infections in residents of long-term care facilities: an agenda for research. Report of an expert panel. *J Am Geriatr Soc* 2002;**50**:570–6.
3. **Gross JS, Neufeld RR, Libow LS, et al.** Autopsy study of the elderly institutionalized patient. Review of 234 autopsies. *Arch Intern Med* 1988;**148**:173–6.
4. **Kerr HD, Byrd JC.** Nursing home patients transferred by ambulance to a VA emergency department. *J Am Geriatr Soc* 1991;**39**:132–6.
5. **Marrie TJ, Durant H, Yates L.** Community-acquired pneumonia requiring hospitalization: 5-year prospective study. *Rev Infect Dis* 1989;**11**:586–99.
6. **Mylotte JM.** Nursing home-acquired pneumonia. *Clin Infect Dis* 2002;**35**:1205–11.
7. **Muder RR.** Pneumonia in residents of long-term care facilities: epidemiology, etiology, management, and prevention. *Am J Med* 1998;**105**:319–30.
8. **Stevenson KB.** Regional data set of infection rates for long-term care facilities: description of a valuable benchmarking tool. *Am J Infect Control* 1999;**27**:20–6.
9. **Lim WS, Macfarlane JT.** A prospective comparison of nursing home acquired pneumonia with community acquired pneumonia. *Eur Respir J* 2001;**18**:362–8.
10. **El Solh AA, Sikka P, Ramadan F, et al.** Etiology of severe pneumonia in the very elderly. *Am J Respir Crit Care Med* 2001;**163**:645–51.
11. **Martinez-Moragon E, Garcia FL, Serra SB, et al.** [Community-acquired pneumonia among the elderly: differences between patients living at home and in nursing homes]. *Arch Bronconeumol* 2004;**40**:547–52.
12. **Niederman MS, Brito V.** Pneumonia in the older patient. *Clin Chest Med* 2007;**28**:751–71, vi.
13. **Donowitz GR, Cox HL.** Bacterial community-acquired pneumonia in older patients. *Clin Geriatr Med* 2007;**23**:515–34, vi.
14. **Fernandez-Sabe N, Carratala J, Roson B, et al.** Community-acquired pneumonia in very elderly patients: causative organisms, clinical characteristics, and outcomes. *Medicine (Baltimore)* 2003;**82**:159–69.
15. **Kollef MH, Shorr A, Tabak YP, et al.** Epidemiology and outcomes of health-care-associated pneumonia: results from a large US database of culture-positive pneumonia. *Chest* 2005;**128**:3854–62.
16. **Micek ST, Kollef KE, Reichley RM, et al.** Health care-associated pneumonia and community-acquired pneumonia: a single-center experience. *Antimicrob Agents Chemother* 2007;**51**:3568–73.
17. **Carratala J, Mykietiak A, Fernandez-Sabe N, et al.** Health care-associated pneumonia requiring hospital admission: epidemiology, antibiotic therapy, and clinical outcomes. *Arch Intern Med* 2007;**167**:1393–9.
18. **Marrie TJ.** Pneumonia in the long-term-care facility. *Infect Control Hosp Epidemiol* 2002;**23**:159–64.
19. **Irvine PW, Van BN, Crossley K.** Causes for hospitalization of nursing home residents: the role of infection. *J Am Geriatr Soc* 1984;**32**:103–7.
20. **El-Solh AA, Aquilina AT, Dhillon RS, et al.** Impact of invasive strategy on management of antimicrobial treatment failure in institutionalized older people with severe pneumonia. *Am J Respir Crit Care Med* 2002;**166**:1038–43.
21. **Tambyah PA, Habib AG, Ng TM, et al.** Community-acquired methicillin-resistant *Staphylococcus aureus* infection in Singapore is usually 'healthcare associated'. *Infect Control Hosp Epidemiol* 2003;**24**:436–8.
22. **Naimi TS, LeDell KH, Como-Sabetti K, et al.** Comparison of community- and health care-associated methicillin-resistant *Staphylococcus aureus* infection. *JAMA* 2003;**290**:2976–84.
23. **Mandell LA, Wunderink RG, Anzueto A, et al.** Infectious Diseases Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. *Clin Infect Dis* 2007;**44** (Suppl 2):S27–72.
24. **Rano A, Agusti C, Jimenez P, et al.** Pulmonary infiltrates in non-HIV immunocompromised patients: a diagnostic approach using non-invasive and bronchoscopic procedures. *Thorax* 2001;**56**:379–87.
25. **Niederman MS, Mandell LA, Anzueto A, et al.** Guidelines for the management of adults with community-acquired pneumonia. Diagnosis, assessment of severity, antimicrobial therapy, and prevention. *Am J Respir Crit Care Med* 2001;**163**:1730–54.
26. **Muder RR, Aghababian RV, Loeb MB, et al.** Nursing home-acquired pneumonia: an emergency department treatment algorithm. *Curr Med Res Opin* 2004;**20**:1309–20.
27. **Furman CD, Rayner AV, Tobin EP.** Pneumonia in older residents of long-term care facilities. *Am Fam Physician* 2004;**70**:1495–500.
28. **Muder RR, Brennen C, Swenson DL, et al.** Pneumonia in a long-term care facility. A prospective study of outcome. *Arch Intern Med* 1996;**156**:2365–70.
29. **Marrie TJ, Blanchard W.** A comparison of nursing home-acquired pneumonia patients with patients with community-acquired pneumonia and nursing home patients without pneumonia. *J Am Geriatr Soc* 1997;**45**:50–5.
30. **Naughton BJ, Mylotte JM, Tayara A.** Outcome of nursing home-acquired pneumonia: derivation and application of a practical model to predict 30 day mortality. *J Am Geriatr Soc* 2000;**48**:1292–9.
31. **Bruto V, Niederman MS.** Healthcare-associated pneumonia is a heterogeneous disease, and all patients do not need the same broad-spectrum antibiotic therapy as complex nosocomial pneumonia. *Curr Opin Infect Dis* 2009;**22**:316–25.
32. **El Solh AA, Pietrantonio C, Bhat A, et al.** Indicators of potentially drug-resistant bacteria in severe nursing home-acquired pneumonia. *Clin Infect Dis* 2004;**39**:474–80.
33. **Dosa D.** Should I hospitalize my resident with nursing home-acquired pneumonia? *J Am Med Dir Assoc* 2006;**7**(3 Suppl):S74–80, 73.
34. **van der Steen JT, Ooms ME, Ader HJ, et al.** Withholding antibiotic treatment in pneumonia patients with dementia: a quantitative observational study. *Arch Intern Med* 2002;**162**:1753–60.
35. **Ruiz M, Ewig S, Marcos MA, et al.** Etiology of community-acquired pneumonia: impact of age, comorbidity, and severity. *Am J Respir Crit Care Med* 1999;**160**:397–405.
36. **Ewig S, Birkner N, Strauss R, et al.** New perspectives on community-acquired pneumonia in 388,406 patients. *Thorax* 2009;**64**:1062–9.
37. **Naughton BJ, Mylotte JM.** Treatment guideline for nursing home-acquired pneumonia based on community practice. *J Am Geriatr Soc* 2000;**48**:82–8.
38. **Venditti M, Falcone M, Corrao S, et al.** Outcomes of patients hospitalized with community-acquired, health care-associated, and hospital-acquired pneumonia. *Ann Intern Med* 2009;**150**:19–26.

REFERENCES

1. **Anderson SD**. Exercise-induced asthma. In: Kay AB, ed. *Allergy and allergic diseases*. Oxford: Blackwell Scientific Publications, 1997:672–711.
2. **Rundell KW**, Im J, Mayers LB, *et al*. Self-reported symptoms and exercise-induced asthma in the elite athlete. *Med Sci Sports Exerc* 2001; **33**:208–13.
3. **Dickinson JW**, Whyte GP, McConnell AK, *et al*. Mid-expiratory flow versus FEV1 measurements in the diagnosis of exercise induced asthma in elite athletes. *Thorax* 2006; **61**:111–4.
4. **Holzer K**, Douglass JA. Exercise induced bronchoconstriction in elite athletes: measuring the fall. *Thorax* 2006; **61**:94–6.
5. **Parsons JP**, Mastrorade JG. Exercise-induced bronchoconstriction in athletes. *Chest* 2005; **128**:3966–74.

Corrections

doi:10.1136/thx.2009.122291corr1

Conway Morris A, Kefala K, Wilkinson TS, *et al*. Diagnostic importance of pulmonary interleukin-1b and interleukin-8 in ventilator-associated pneumonia. *Thorax* 2010; **65**:201–7. This article should have included the note that Dr Kefala was joint first author.

doi:10.1136/thx.2009.124776corr1

Polverino E, Dambava P, Cilloniz C, *et al*. Nursing home-acquired pneumonia: a 10 year single-centre experience. *Thorax* 2010; **65**:354–59. The correct affiliation for affiliation 1 should have read “Respiratory Department, Hospital Clinic-IDIBAPS, Barcelona-Spain, Centro de Investigación Biomedica En Red-Enfermedades Respiratorias (CibeRes, CB06/06/0028, el Ciberes es una iniciativa del ISCIII) – 2009SGRQ - <http://www.idibapsrespiratoryresearch.org>.”

doi:10.1136/thx.2009.133108corr1

Millett C, Glantz SA. Assigning an ‘18’ rating to movies with tobacco imagery is essential to reduce youth smoking. *Thorax* 2010; **65**:377–8. The authors referred to a paper by McNeil *et al*; this should have been Lyons *et al* (Lyons A, McNeill A, Chen Y, *et al*).

doi:10.1136/thx.2009.130716corr1

Lyons A, McNeill A, Chen Y, *et al*. Tobacco and tobacco branding in films most popular in the UK from 1989 to 2008. *Thorax* 2010; **65**:417–22. There is an error in figure legend 2 which currently reads “Trends in all tobacco intervals and tobacco use intervals per hour per **day** by British Board of Film Classification (BBFC) category (all figures expressed as means).” It should have read: “Trends in all tobacco intervals and tobacco use intervals per hour per **year** by British Board of Film Classification (BBFC) category (all figures expressed as means).”

doi:10.1136/thx.2009.127274corr1

Kemp SV, El Batrawy SH, Harrison RN, *et al*. Learning curves for endobronchial ultrasound using cusum analysis. *Thorax* 2010; **65**:534–8. The author name A Roselli should have read A Rosell.