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Community Acquired Pneumonia - Definitions, Epidemiology And Risk Factors

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Abstract

Community-acquired pneumonia (CAP) refers to an infectious process resulting from the invasion and overgrowth of microorganisms in lung parenchyma in a non-hospitalized population. Occurrence of respiratory symptoms or signs in the presence of a chest infiltrate is essential for the diagnosis of CAP. CAP occurs in all age groups but children less than 5 years, the elderly and those with underlying risk factor(s) are particularly susceptible. CAP may be classified and managed by morphological patterns, etiological organism and empirical 'host-organism pattern' approach. Host factors play an important role in determining the severity of CAP and certain risk factors are associated with specific disease patterns and specific microorganisms.

Key Words: Community-acquired pneumonia, risk factor, epidemiology, etiology, micro-organism

Definitions

Pneumonia is a heterogeneous entity defined on the basis of the situation in which it is diagnosed, the etiological organism and co-morbidity in the host. Pneumonia is commonly classified as community- acquired or nosocomial (hospital acquired),

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depending on the environmental setting in which it is acquired. Ventilator associated pneumonia is a specific type of nosocomial pneumonia seen in the intensive care unit. Pneumonia may occur in healthy individuals or in those who are immunosuppressed or have a variety of comorbidities and risk factors. Community-acquired pneumonia (CAP) refers to an infectious process resulting from the invasion and overgrowth of microorganisms in lung parenchyma in a non-hospitalized population. Occurrence of respiratory symptoms or signs in the presence of a chest infiltrate is essential for the diagnosis of CAP (1). Lung infiltrates are sometimes not evident on frontal radiography but computed tomography which may reveal such occult infiltrates is seldom required for routine diagnosis (2). On the contrary, pneumonia occurring 48 hours or more after admission, and not incubating at the time of admission is referred to as hospital-acquired pneumonia (HAP). Pneumonia arising more than 48-72 hours after endotracheal intubation is called ventilator-associated pneumonia (VAP).

CAP may be approached by morphological patterns, etiological organism and empirical 'host-organism pattern' approach. Etiologically, the most common bacterial causes of CAP include *Streptococcus pneumoniae,* and *H. influenza, Brahmanella Catarrhalis, Mycoplasma pneumoniae, Chlamydia pneumoniae, and Legionella pneumophila*. The last 3 organisms are referred to as *atypical* pathogens. Enteric gram-negatives, *Staphylococcus aureus,* anaerobes, *Ps. Aeruginosa,* viruses, mycobacteria and fungi are unusual causes of CAP and constitute less than 5% of cases together.

The diagnosis is often clinical. A retrospective study found that ICD-10 codes had only moderate sensitivity for diagnosis (3). The accuracy of coding data to identify laboratory confirmed pneumococcal pneumonia depends upon case-definitions. Therefore to maximize the sensitivity of diagnosing by medical chart review, it is important to use a combination of many ICD-9-CM codes (4).

CAP is also defined on the basis of its response to therapy. Some of these terms which are commonly used but imprecisely defined include 'progressive pneumonia', 'slowly-resolving' or 'non-responding pneumonia' and 'non-resolving pneumonia'. With appropriate antibiotics, resolution of symptoms begins at 48-72 hours. The lack of complete radiological resolution at 12 weeks is described as 'non-resolving pneumonia' (5, 6).

Epidemiology

CAP is an important cause of morbidity and mortality world-wide. In the United States, pneumonia is the sixth leading cause of death. The disease affects all age groups but particularly the children under the age of 5 years, the elderly and those with an underlying risk factor. The annual incidence in those aged above 65 years is estimated between 25 and 55 cases per 1000 while the overall annual incidence of CAP is about 2%. In the U.S., the overall attack rate is about 12 cases per 1000 persons per year. The incidence is also high in people living in old-age homes or other similar institutions.

The prevalence is reported as high in the developing countries. Of 15 million deaths in children from acute respiratory infections worldwide, about a third result from pneumonia. This is attributable to both the environmental and the host risk factors. There are limited data on the epidemiology and population prevalence of CAP in India. Whatever data is reported is drawn from the hospitalized patients (7). A few other hospital based studies are also reported on clinical and/or microbiological features of atypical forms of CAPs, such as the Mycoplasma and Chlamydia pneumonia especially in children (8-10). Mycoplasma pneumonia was reported in 27.4% of 62 children admitted with CAP in a one year period in Delhi – only 4 (6.4%) were reported as seropositive for Chlamydia (11).

Streptococcus pneumoniae remains the most common cause of CAP in India as anywhere else in the world. In a multicentre hospital surveillance for *Strep pneumoniae*, in six hospitals in India over a period of 4 years, there were 3686 cases with suspected pneumonia and the clinical syndrome of pneumonia was identified in 93 out of 314 cases (29.6%) with invasive pneumococcal disease (12). A 19% case fatality of pneumonia was also described (12). In our own analysis of 167 patients of CAP admitted in the Respiratory Intensive Care Unit (RICU) over a 7-year period (Fig 1), 68 (40.7%) had died after an average hospital stay of 8 days (unpublished data).

Causes and Risk Factors

The pathogenesis of CAP involves an imbalance between the pathogen's invasiveness and the host immunity. What Louis Pasteur had said more than a hundred years ago about the host factors, is revealing: "The germ is nothing; the soil is everything". In general, there are numerous host factors which predispose to CAP. While the virulence of the microorganism

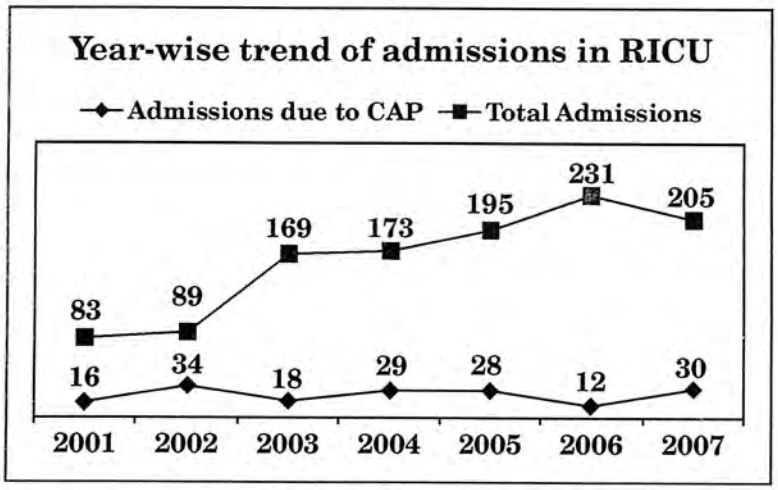

Figure 1: Year-wise number of patients with CAP against total admissions in the RICU at the PGIMER, Chandigarh

depending upon the group, the strain and the type is the root cause of infection, the host factors determine the occurrence and the severity (13). Infants, young children and the elderly are the most vulnerable. There are several predisposing and disease modifying factors which include the presence of a systemic illness, a behavioral problem or an extraneous environmental exposure (Table 1).

Table 1: Important host factors responsible for CAP

A.	Pre-disposing factors: Chronic Obstructive Pulmonary Disease, Diabetes mellitus, congestive heart failure, coronary artery disease, malignancies, neurological, renal or liver disease.
B.	Modifying factors: include those factors which increase the risk of infection with drug resistance and unusual pathogens:
	a. Age > 65 years
	b. β-Lactam therapy within past 3 months
	c. Immunosuppression
	d. Multiple medical co-morbidities
	e. Alcoholism
	f. Exposure to children in day care centres

Table 2: Risk factors linked to CAP with less common microorganisms

A. Enteric gram negative infection
   a. Recent antibiotic therapy
   b. Underlying cardiopulmonary disease
   c. Residence in a nursing home
   d. Multiple medical co morbiditics
B. *Ps.aeruginosa* infection
   a. Structural lung disease
   b. Antibiotic therapy for > 7 days in the past month
   c. Corticosteroids (at least 10 mg prednisolone/day)
   d. Malnutrition
C. Drug-resistant *Streptococcus* pneumonia (DRSP)
   a. Sicker inpatient population
   b. Immuno-compromised patients
   c. New procedures & instrumentation
   d. Emerging pathogens
   e. Complacency regarding antibiotics
   f. Ineffective infection control and compliance
   g. Increased antibiotic use
D. *Staph aureus* pneumonia
   a. Advanced age
   b. Prolonged hospitalization
   c. Underlying lung disease
   d. Prior antibiotic therapy
E. Anaerobic bacterial pneumonia
   a. Aspiration
   b. Presence of gingivitis
   c. Bronchopleural fistula
   d. Pneumonia complicating bronchial obstruction

Some risk factors are also identifiable for specific types of CAPs caused by different microorganisms. For example specific risk factors for pneumococcal infections include dementia, seizures, HIV infection and systemic illnesses such as the chronic obstructive pulmonary disease (COPD), congestive heart failure (CHF) and cerebrovascular disease (14, 15). *Haemophilus influenzae* is most common in patients with an underlying COPD and the use of antibiotics or oral corticosteroids within the past 3 months (16, 17). The risk factors for some of the relatively uncommon CAPs such as those caused by pseudomonas, *Staph aureus*, gram negative bacteria, drug resistant *Streptococcus pneumoniae* (DRSP) or anaerobic organisms are generally identifiable (Table 2) (18-22).

Immunosuppression with drugs or HIV infection is an important risk factor on the rise in the recent years. *Strep pneumoniae* and *H. influenzae* are the common organisms in the developed as well as the developing world (23, 24). Such patients are also likely to develop CAPs with different atypical organisms including the mycobacteria, the viruses (such as the cytomegalovirus, herpes simplex, influenza virus and respiratory syncytial virus) and the fungi (25, 26). In a recently published study from Chennai in South India on 200 HIV-positive adults with CAP and 75 HIV-negative adults with CAP, the prevalence of *M. pneumoniae* in HIV-positive patients was higher (17%) by induced sputum examination (27). The sero-prevalence of anti-*M. pneumoniae* Ig M was 11.7% and 14.3% respectively by ELISA and gelatin micro-particle agglutination test (27) in the two groups.

Tuberculosis is another important respiratory infection in HIV positive patients which may present with pulmonary infiltrates and clinical features of CAP (28-30). Tuberculosis therefore requires to be kept in the differential diagnosis of CAP in all immuno-suppressed patients especially those with HIV infection.

Conclusions

CAP remains an important cause of morbidity and mortality world-over. Diagnosis is often clinical, although radiologic confirmation is desirable, if readily available. Data on the epidemiology and population prevalence of CAP from India, despite being limited, suggest that pneumococcus remains the most common etiological agent. It is important to identify risk factors and other host factors in patients with CAP since these are known to influence the responsible etiological agents as well as presentation and course of the disease.

References

1. Bernstein JM (1999). Treatment of community-acquired pneumonia – IDSA guidelines. Infectious Diseases Society of America. *Chest* **115**:9S-13S.
2. Basi SK, Marrie TJ, Huang JQ, *et al* (2004). Patients admitted to hospital with suspected pneumonia and normal chest radiographs: epidemiology, microbiology and outcomes. *Am J Med* **117**:305-11.
3. Van de Garde EM, Oosterheert JJ, Bonten M, *et al* (2007). International classification of diseases codes showed modest sensitivity for detecting community-acquired pneumonia. *J Clin Epidemiol* **60**:834-38.
4. Guevara RE, Butler JC, Marston BJ, Plouffe JF, File TM Jr, Breiman RF (1999). Accuracy of ICD-9-CM codes in detecting community-acquired pneumococcal pneumonia for incidence and vaccine efficacy studies. *Am J Epidemiol* **149**:282-9.
5. Cunha BA (2000). Slowly resolving and nonresolving pneumonias. *Drugs Today (Barc)* **36**:829-34.
6. Kuru T, Lynch JP 3rd (1999). Nonresolving or slowing resolving pneumonia. *Clin Chest Med* **20**:623-51.
7. Bansal S, Kashyap S, Pal LS, Goel A (2004). Clinical and bacteriological profile of community acquired pneumonia in Shimla, Himachal Pradesh. *Indian J Chest Dis Allied Sci* **46**:17-22.
8. Dey AB, Chaudhry R, Kumar P, Nisar N, Nagarkar KM (2000). Mycoplasma pneumoniae and community-acquired pneumonia. *Natl Med J India* **13**:66-70.
9. Jain R, Jain A, Agarwal J, Awasthi S (2007). Chlamydia sp. in hospitalized children with community-acquired pneumonia. *Indian Pediatr* **44**:216-8.
10. Chugh K (1999). Pneumonia due to unusual organisms in children. *Indian J Pediatr* **66**:929-36.
11. Chaudhry R, Nazima N, Dhawan B, Kabra SK (1998). Prevalence of Mycoplasma pneumoniae and Chlamydia pneumoniae in children with community-acquired pneumonia. *Indian J Pediatr* **65**:717-21.
12. Prospective multicentre hospital surveillance of Streptococcus pneumoniae disease in India. Invasive Bacterial Infection Surveillance (IBIS) Group,

International Clinical Epidemiology Network (INCLEN) (1999). *Lancet* **353**:1216-21.

13. Apisarnthanarak A, Linda MM (2005). Etiology of community-acquired pneumonia. *Clin Chest Med* **26**:47-55.
14. Hoge CW, Reichler MR, Dominguez EA, *et al* (1994). An epidemic of pneumococcal disease in an over-crowded, inadequately ventilated jail. *N Engl J Med* **331**:643-8.
15. Nuorti JP, Butler JC, Farley MM, *et al* (2000). Cigarette smoking and invasive pneumococcal disease. *N Engl J Med* **342**:681-9.
16. Torres A, Dorca J, Zalacain R, *et al* (1996). Community-acquired pneumonia in chronic obstructive pulmonary disease: a Spanish multicenter study. *Am J Respir Crit Care Med* **154**:1456-61.
17. Medeiros AA (1998). Comparison of oral cephalosporins and their activities against β-lactamase producing bacteria. *Infect Dis Clin Pract* **7**:S272-9.
18. Cross JT, Campbell JR GD (1999). Drug-resistant pathogens in community and hospital-acquired pneumonia. *Clin Chest Med* **20**:499-506.
19. Al-Ujayli B, Nafziger DA, Saravolatz L (1995). Pneumonia due to *Staphylococcus aureus* infection. *Clin Chest Med* **16**:111-20.
20. Hatchette TF, Gupta R, Marrie TJ (2000). *Pseudomonas aeruginosa* community-acquired pneumonia in previously healthy adults: case report and review of the literature. *Clin Infect Dis* **31**:1349-56.
21. Bartlett JG (1993). Anaerobic bacterial infections of the lung and pleural space. *Clin Infect Dis* **16**:S248-55.
22. Iwahara T, Ichiyama S, Nada T, *et al* (1994). Clinical and epidemiologic investigations of nosocomial pulmonary infections caused by methicillin-resistant Staphylococcus aureus. *Chest* **105**:826-31.
23. Rimland D, Navin TR, Lennox JL, Jernigan JA, Kaplan J, Erdman D, *et al* (2002). Prospective study of etiologic agents of community-acquired pneumonia in patients with HIV infection. *AIDS (London, England)* **16**:85-95.
24. Scott JA, Hall AJ, Muyodi C, Lowe B, Ross M, Chohan B, *et al* (2000). Aetiology, outcome and risk factors for mortality among adults with

acute pneumonia in Kenya. *Lancet* **355**:1225-30.

25. Park DR, Sherbin VL, Goodman MS, *et al* (2001). The etiology of community-acquired pneumonia at an urban public hospital: influence of human immunodeficiency virus infection and initial severity of illness. *Clin Infect Dis* **184**:268-77.

26. Navin TR, Rimland D, Lennox JL, *et al* (2000). Risk factors for community-acquired pneumonia among persons infected with human immunodeficiency virus. *Clin Infect Dis* **181**:158-64.

27. Shankar EM, Kumarasamy N, Vignesh R, *et al* (2007). Epidemiological studies on pulmonary pathogens in HIV-positive and negative subjects with or without community-acquired pneumonia with special emphasis on Mycoplasma pneumoniae. *Jpn J Infect Dis* **60**:337-41.

28. Havlir DV, Barnes PF (1999). Tuberculosis in patients with human immunodeficiency virus infection. *N Engl J Med* **340**:367-73.

29. Aaron L, Saadoun D, Calatroni I, Launay O, Memain N, Vincent V, *et al* (2004). Tuberculosis in HIV-infected patients: a comprehensive review. *Clin Microbiol Infect* **10**:388-98.

30. Corbett EL, Marston B, Churchyard GJ, DeCock KM (2006). Tuberculosis in sub-Saharan Africa: opportunities, challenges and change in the era of antiretroviral treatment. *Lancet* **367**:926-37.