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Pneumonia: how important are local epidemiology and smoking habits?

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In this issue of *Monaldi Archives*, Al-Muhairi *et al*. describe the epidemiology of Community- and Hospital-acquired pneumonia in United Arab Emirates over five years [1].

Most of the cases (287, 80%) were actually community-acquired (CAP) and only 74 hospital acquired (HAP). Considering the five-year observation only a few HAP cases each year were observed and no clear conclusion can be drawn from this series.

More interesting are the observations that the annual admission rate for CAP increased from 250/100,000 admissions in 1997 to 710/100,000 in 2002 and the presence of a seasonal trend in the admission rate.

The lack of any established rules for hospital admission can only partially explain the increase in the number of patients hospitalised. A change in population risk factors or in CAP etiology can also be involved but, unfortunately, these variables are not addressed by this study.

For example, in Al-Muhairi study, as commonly reported in the majority of studies into pneumonia, there is a large proportion of cases that have no pathogen identified, mainly because the appropriate tests were not performed [2]. In this study less than 30% of patients had sputum examination, less than 50% blood culture and only 14/287 had serology tests. This is a major pitfall of the study, that cannot give any indication on CAP etiology in the examined aerea.

The seasonal differences in CAP incidence seem to be clearly related to population migration. This is an interesting point, rarely addressed in literature. The authors suggest that the peak of pneumonia cases follows the return from Asia of the expatriate population, this means a transmission of infections from Asian residents to local population. What is not clear is that only 3% of admitted patients are Asians. This may imply a high rate of infection spreading between Asian immigrants and the local population, underlying the importance of etiologic studies in this setting. Both increasing incidence and seasonal variation should foster the authors to design a new study to better investigate the epidemiological risk factors and the etiology of CAP in their population.

Another interesting article in this issue of Monaldi Archives addresses the effect of cigarette smoking on hydrogen peroxide (H_2O_2) and thiobarbituric reactive substances (TBARs) concentrations in exhaled breath condensate (EBC) in patients with CAP [3].

The effects of tobacco smoke on the airway mucosa are well known. The chronic inhalation of smoke favours the adhesion of *S. pneumoniae* and *H. influenzae* to the buccal epithelium. Smoking alters mucociliary transport, humoral and cellular defences, and epithelial cells. The proportion of smokers in hospitalised pneumonias is generally increased, which would indicate a role of smoking in the acquisition of infection.

In the Stolarek *et al*. study the different responses to oxidative stress in smokers and non-smokers with CAP is addressed. The study relies on the use of EBC, an interesting non-invasive technique for obtaining samples from the lungs. In EBC large number of mediators including hydrogen peroxide, isoprostanes, leukotrienes, nitrogen oxides, peptides and cytokines can be detected. Data from literature shows that concentrations of these mediators are influenced by lung diseases and modulated by therapeutic interventions.

As recently stated in the ATS/ERS recommendations on EBC [4] H_2O_2 concentrations increase during bronchopulmonary infections and decrease after antibiotic treatment in cystic fibrosis patients [5].

However, day to day intra-subject coefficient of variation of 43% in healthy subjects has been reported [6].

The effects of smoking on EBC mediators concentration is well known; smoking has considerable effect on H_2O_2, isoprostane, nitrite and nitrotyrosine levels [7-9].

In Stolarek *et al*. [3] study the H_2O_2 time-related concentrations detected in EBC result lower in smokers than in non-smokers, whereas TBARs levels increase more in smokers than in non-smokers. These surprising results are probably related, as stated by the authors, to an impaired host response to infection related to a smoking-sustained airways chronic inflammation.

Clearly more studies are needed to ascertain the pathogenetic role of these findings and the possible need for different treatment approaches to smokers with pneumonia.

In conclusion, this issue of *Monaldi Archives* addresses two important problems.

As stated by all guidelines local epidemiology knowledge is of primary importance for correct management of lower respiratory tract infections and the article analysed [1] is a good example of the problems that are faced when obtaining valid epidemiologic data. However risk factor identification is clearly mandatory for improving treatment of pneumonia patients. Smoking effects on oxidative response is one of the potentially important targets concerning new approaches to the management of acute and chronic airway inflammation. Stolarek *et al.* [3] study results give us more questions than answers, indicating the need for a better understanding of the pathophysiology of airway response to infections.

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