Atypical pneumonias in adults

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The atypical pneumonia syndrome is a clinical entity caused by a diverse spectrum of microorganisms. It should be differentiated from pneumococcal pneumonia, as empirical therapy differs significantly.

Introduction

The atypical pneumonia syndrome is a distinct form of community-acquired pneumonia (CAP) characterized by the subacute onset of symptoms and a prominence of nonspecific *systemic manifestations*. *Extrapulmonary symptoms initially predominate* (fever, headache, athralgia, myalgia and gastrointestinal symptoms), with relative *limited findings* on *respiratory examination* (commonly only crackles). The most prominent early respiratory symptom is *cough*, with initially scant and mucoid sputum. This may become purulent as the disease progresses. Dyspnoea and pleuritic chest pains are rare. A low-grade leucocytosis is usually present and biochemical and haematological investigations may be abnormal (see below). The CXR findings can range from unremarkable to interstitial reticulo-nodular patterns to even more typical lobar and bronchopneumonic infiltrates.

There is uncertainty about the true incidence of atypical pneumonia in South Africa, but differentiating this clinical entity from the "typical" (mostly pneumococcal) CAP has important therapeutic implications, as these organisms as a rule are not sensitive to beta lactam antibiotics.

Although the aetiologies of atypical pneumonia are numerous, certain agents still make out the largest percentage in southern Africa.

Mycoplasma pneumoniae is a common cause of mild lobar pneumonia in a young adult, but frequently causes other lower and upper respiratory infections. Only rarely does haemolitic anaemia, erythema multiforme, bullous myringitis, encephalitis, transverse myelitis, myocarditis and other sequelae complicate this picture. A skin rash (mostly maculo-papular) is present in 15%.

Chlamydia pneumoniae (strain TWAR) causes pneumonia resembling *M. pneumoniae*.

Evidence suggests the prevalence of ***Legionella pneumophila*** to be low in South Africa. The spectrum of pathology caused by the pathogen ranges from a mild benign influenza-like illness to a full-blown atypical pneumonia with prominent extrapulmonary affliction, including a deterioration in mental state, severe abdominal complaints, renal and hepatic dysfunction and severe hyponatraemia.

Several **zoonoses** are associated with atypical pneumonia. These include *Chlamydia psittaci, Coxiella burnetti and Francisella tularences*.

The syndrome of atypical pneumonia is not limited to bacteria. **Mycobacteria**, several **viruses** (RSV, measles, VZV, etc.) and even **fungi** are also implicated. *Pneumocystis carinii* causes the classical atypical picture in the immunocompromised host.

Treatment/management protocol

The **diagnosis** of atypical pneumonia is largely **clinical**. Most of the organisms mentioned are not amendable to culture (or require special media). **Serology** therefore plays an important role in making a definitive diagnosis, although this is not always critical. One acceptable approach would be to obtain a *baseline sample* of serum and to store this specimen. As a single antibody titre is difficult to interpret, this specimen only requires initial evaluation in the critically ill patient or where empirical therapy fails. A *repeat specimen* can be taken *two weeks later* and both these samples can then be analysed if aetiology is still uncertain. The sensitivity and specificity of serological markers vary extensively, but as a general rule a fourfold rise in antibody titre is diagnostic as the specific cause. It is suggested that the clinician discusses each case with the laboratory involved, to prevent being misled by insignificant titres or different techniques used in these laboratories. **Sputum** should be stained for acid-fast *bacilli* where pulmonary TB is suspected, and for P. carinii if appropriate. Gram stains and culture on respiratory secretions are performed in all severe infections. **Certain laboratory findings** may point in a certain direction (e.g. cold haemagglutinins in the case of *mycoplasma*, hyponatraemia with *Leigonella* and a raised LDH with *Pneumocystis*), but none of these are diagnostic *per se*.

General therapeutic and supportive measures are as for pneumococcal pneumonia.

Antibiotic therapy is initially *empirically* aimed at the commonest aetiological agents and should include a *macrolide antibiotic* as first-line therapy. Traditionally Erythromycin was used extensively, but the newer generation of macrolides (Clarithro-, Roxithro- and Azithromycin) has proven to be clinically effective with a superior pharmacokinetic and safety profile. Alternatives include the *tetracyclines* and the *newer fluoroquinolones* (moxifloxacin, etc.).

Current guidelines, however, do not recommend these antibiotics as monotherapy in cases of suspected pneumococcal pneumonia (exception: moxifloxacin). It may thus be appropriate to use combination therapy (i.e. a macrolide and a beta-lactam antibiotic or alternative) in cases where accurate differentiation is impossible.

This empirical approach covers more than 90% of cases, but certain agents require different therapy, *M. tuberculosis* being but one example.

References

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