LETTERS TO THE EDITOR

BTS guidelines on CAP

The new BTS guidelines on the management of community-acquired pneumonia (CAP) in adults are welcome if they lead to improved diagnosis of pneumonia, better assessment of severity of illness, and thus more appropriate treatment according to clinical needs. It is widely accepted, however, that inappropriate implementation of the previous guideline contributed to large increases in unnecessary use of broad spectrum antibiotics with resultant increases in antibiotic resistance and *Streptococcus pneumoniae* difficult infection. The authors acknowledge this, but the new guidelines seem likely to continue this unfortunate trend.

Firstly, there is no mention of the use of oral penicillin for treatment of mild cases of CAP. This is a first line choice in Scandinavian countries where this have a commendably restrained history of antibiotic use (and consequently low rates of resistance). The new BTS guideline recommendation for widespread use of the broader spectrum amoxicillin cannot help current antibiotic resistance problems. The pharmacodynamic arguments favouring amoxicillin may be important in those areas having problems with penicillin intermediate and resistant pneumococci, but in many areas of the UK—including much of Scotland—these strains are rare. Did the authors consider oral penicillin as an option for mild cases?

Secondly, for treatment of severe pneumonia there is no mention of parenteral penicillin. The recommendation of co-amoxiclav or cephradine for this condition, while covering uncommon Gram negative pathogens and mexitillin sensitive *Staphylococcus aureus* (MSSA), may lead to inadequate treatment of CAP due to penicillin resistant pneumococci. Surely benzyl penicillin is an option in young previously healthy people with severe CAP (the majority of whom will have pneumococcal infection). Then, if there is a reasonable risk of infection with a pneumococcus with reduced susceptibility to penicillin, the choice of benzyl penicillin can be raised accordingly.

Thirdly, the recommendations for macrolide use in the first version of the guideline have probably been the main reason for the doubling of macrolide consumption in our local hospital since the previous guidelines were introduced (unpublished observation). If this observation is indicative of a more widespread trend, it may well be contributing to the current national problem with MRSA and other macrolide resistant organisms. To what benefit I wonder? Certainly, a laboratory diagnosis of atypical pneumonia is rare in our population. Isn’t this another case for stratifying patients according to risk rather than treating all severely ill hospitalised patients with a macrolide?

I appreciate the huge body of evidence considered by the authors and the disappointing number of studies which were helpful in guiding best recommendations for treatment. Nevertheless, at a time when there is widespread concern about inappropriate antibiotic use, much of it with broad spectrum agents, it is crucial that new guidelines urge restrained prescribing unless the risks (inadequate spectrum clearly) clearly outweigh the benefits (reduced ecological damage). At the same time, severe cases require the best treatment and this should not be compromised out of a desire to do the impossible and cover all conceivable (but unlikely) pathogens all of the time.

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References


Transudates and exudates

Joseph et al have made a valuable contribution to the evaluation of pleural effusions. However, we would like to sound a note of caution. Throughout the literature, including the study by Joseph and others, there remains the same: no single test is diagnostic for transudates or exudates. Thus, reliance on such a test can be misleading and lead to either under or over investigation.

Rarely in the literature is there any discussion regarding the place of pleural fluid protein or lactate dehydrogenase (LDH) estimation. Specifically, how does it alter management? Does the finding of a transudate obviate the need for further investigation? The main problem is that a significant number of malignant effusions are classified as transudates, whichever method is used. The cause of a transudate is usually clinically obvious. If, however, there is no obvious underlying cause, surely cytological and/or histological examination should still be sought, as for an exudate.

Estimation of pleural fluid protein or LDH is also irrelevant if the fluid is bloodstained, as here further investigation for possible malignancy is warranted anyway.

We propose that the principal use for pleural fluid protein or LDH measurement is when a probable underlying cause for a transudative effusion is apparent, such as heart failure or hypoalbuminaemia, and the fluid is not bloodstained. In this situation the finding of a transudate may help to reassure that no further investigation is necessary except observation, and that a trial of treatment with, for example, diuretics may be of help.

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Authors’ reply

We appreciate the comments by Quantrill and Dabal on our recent paper and would like to clarify the issues raised by them. By definition, when the pleural fluid is classified as a transudate, it indicates that a pathological process does not involve the pleural surface and that an effusion is formed because of a hydrostatic imbalance. If the pleural fluid is bloodstained, it therefore suggests disruption of the pleural membrane by an inflammatory or malignant process and hence cannot be classified as a transudate, which obviates the need for estimation of fluid LDH or protein estimation for diagnostic classification. However, as suggested by Quantrill and Dabal, an occasional malignancy may present as a transudate, in which case the mechanism is usually an effusion from collapse of a lobe causing an increase in the negative pleural pressure. Whatever the mechanism, if clinical suspicion for malignancy is high, further appropriate investigations need to be carried out.

Furthermore, Quantrill and Dabal state that hypoalbuminaemia is an apparent cause for transudative effusions. However, recent literature shows that hypoalbuminaemia per se may not cause pleural effusions. In our paper we have provided the positive likelihood ratios of the various tests so a clinician armed with the pretest probability for any individual patient and the positive likelihood ratio can work out the post-test probability using a standard nomogram. In light of the above, we suggest that fluid LDH and total protein ratio are useful in the diagnostic separation of pleural effusions.

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References

1 Joseph J, Badrinath P, Basran GS, et al. Is the pleural fluid transudate or exudate? A probable underlying cause for a transudative effusion is apparent, such as heart failure or hypoalbuminaemia, and the fluid is not bloodstained. In this situation the finding of a transudate may help to reassure that no further investigation is necessary except observation, and that a trial of treatment with, for example, diuretics may be of help.

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References

1 Joseph J, Badrinath P, Basran GS, et al. Is the pleural fluid transudate or exudate? A
infection manifesting in an HIV infected
et al.
We read with interest the report by Bach-
health interventions for tuberculosis cases.
as a presumed case of tuberculosis, it is
not have differed had the patient been treated
despite the increase in CD4 cell count cannot
ent negativity of tuberculin skin testing (TST)
to growth in hospital hot water systems.
associated with nosocomial outbreaks related
diagnosis.
Too little is known about other NTM (such as
M avium
Mycobacterium xenopi pulmonary infection in
1
Bachmeyer C
et al
Keddissi JI, Kinasewitz GT.
Hypoalbuninemia as a cause of pleural effusion in
3
4
5
6

Authors’ reply
We thank Drs Salazar-Schicchi and Nachman for their interest in our paper and their valuable comments. However, we consider that Mycobacterium xenopi was responsible for the patient’s disease despite the fact that the microbiological diagnosis was not “defini-
tive”. Indeed, the criteria of the American Thoracic Society were not fulfilled.1 These cri-
dria do not seem to be applicable to M xenopi in HIV infected patients, in whom two positive cultures of M xenopi and no other cause of symptoms have been proposed as criteria for the diagnosis.2 Our patient also did not fulfil these criteria. Furthermore, we were concerned about the possible role of other pathogens—especially M tuberculosis—since coexistent pul-
monary infections due to other pathogens had been reported.3 However, no other pathogens were found and a search for M tuberculosis in the three sputum samples and broncho-
alveolar lavage fluid was negative on direct microscopy examination and culture. This is rare in cavitary tuberculosis and makes this diagnosis unlikely.
Mycobacterium xenopi may be found in hospital water taps, hot water storage tanks, and contaminated bronchoscopes.4 Environmental contamination seemed unlikely since M xenopi was not isolated from samples in the microbiology laboratory during the period of management of our patient.
We conclude that M xenopi can be the cause of a lung disease in HIV infected patients that resembles tuberculosis and clinicians should not disregard the significance of this organism when isolated from respiratory specimen, even from only one.

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References

Pharmacology of Asthma
A course on “Pharmacology of Asthma”
Critical care training in Spain

In the review entitled “The pulmonary physi-
critical care?” by M J D Griffiths and T W Evans which appeared in the January issue of
Thorax (2002; 57:77–8), it was incorrectly
stated that: “In Spain 4 years of training are
required to achieve specialist status, 2 years of
which are in intensive care medicine”. This
should have read: “In Spain 5 years of training
are required to achieve specialist status, 3
years of which are in intensive care medi-
cine”.

Low dose of inhaled steroids
and prevention of asthma death

In the paper by J C Kips and R A Pauwels
entitled “Low dose inhaled corticosteroids and
the prevention of death from asthma” which appeared in the 2001 Year in Review
published as Supplement II in September
2001 (Thorax 2001; 56(Suppl II):ii74–ii78), an
error occurred in the abstract of the Introduc-
tory article by Suissa et al (N Engl J Med
2000;343:332–6). In the Results section it is
stated that “...the rate of death from asthma
declined by 2% with each additional canister of
inhaled corticosteroids used in the previous
year ...”. This should have read “... the rate of
death from asthma decreased by 21% with
each additional canister of inhaled cortico-
steroids used in the previous year ...”. The
publishers apologise for this error.
Transudates and exudates

S J Quantrill and L Dabal

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