Allergic bronchopulmonary aspergillosis

Sir,

Brueton et al. reported the occurrence of 7 cases of allergic bronchopulmonary aspergillosis in patients with cystic fibrosis (CF) in a short period of time, for which they could find no explanation; they also mentioned that asymptomatic patients in children with CF. We were interested in the occurrence of Aspergillus fumigatus being isolated more often at this hospital we did not know whether this reflected improved microbiology techniques or a genuine increase. Investigation of possible reasons included an examination of nebulisers, as many patients with CF required nebulised drugs.

One machine showed a collection of fluff on the air intake grill. Tests showed that the machine discharged large numbers of A. fumigatus from the air supply to the nebuliser, and positive cultures for A. fumigatus were obtained too from fluff taken from within the casing, the inlet filter, and from both sides of the outlet filter. We feel that the design of this particular machine is unsatisfactory because of the siting of the air inlet.

Brueton et al. commented that the timing of symptoms correlated with seasons previously reported to have a high atmospheric count of aspergilli. The discharge of spores from the nebuliser must be presumed to act in a similar manner. Also, they questioned whether the increased incidence of atopy might reflect some aspect of treatment. We have no evidence that nebulisers initiate allergic bronchopulmonary aspergillosis but they must exacerbate the problem. Our machine was one of 2 machines used by inpatients with CF. Although we examined the second machine, and a number of others of different designs, we did not find A. fumigatus. However, it was noticed that the nebuliser into which the drug is placed is sometimes inadequately cleaned in machines used in the home. It is possible that aspergilli, and perhaps other bacteria, are unwittingly being nebulised into patients. We feel that further studies are needed to determine the extent of this problem.

References


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Professor Anderson comments:

I thank Dr George and Dr Gillett for their letter on their findings concerning inhalation machines and the presence of Aspergillus fumigatus; we asked them to make such a

Dr Pearson comments:

The use of the UCH figures as an absolute standard but if the Salisbury figures are held constant (as in our Table) there is no significant difference by the χ² test using Yates’s correction (as Pearson did) between 19 deaths (47.5% mortality) and 38 deaths (95% mortality) at UCH.

No amount of mathematical manipulation will alter the fact that there is no statistically significant difference in mortality between Salisbury and UCH on the quoted figures. We therefore cannot accept Pearson’s conclusion that the small size of the Salisbury sample alone makes comparative merits of the methods of care impossible from the data supplied. A controlled trial, preferably one that includes morbidity as well as mortality, is the only way to see which method of care is preferable statistically.

References


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Table Comparison of different possible levels of mortality at UCH with the observed mortality at Salisbury

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<th>Lived</th>
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<td>Hypothetical mortality of 47.5% Salisbury</td>
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<td>UCH</td>
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<td>χ² = 3.536, P &gt; 0.05</td>
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<td>χ² = 3.123, P &gt; 0.05</td>
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Dr Pearson comments:

The use of the UCH figures as a standard with which to compare the results of neonatal care at Salisbury was not my idea but that of Dr Hughes-Davies. I tried to suggest that this comparison is invalid if such small numbers are involved. Dr Burman and Dr Morris appear to be agreeing with this conclusion. Had the UCH team compared their results with those obtained at Salisbury and drawn conclusions from such a comparison, the approach adopted by Burman and Morris would have shown the inaccuracies inherent in such conclusions in exactly the same way.

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Professor Anderson comments:

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