persistent bacterial bronchitis/persistent endobronchial infection. This is indeed the case in an adult with dry sputum who would be reflexively given a clear history of expectorating yellow/green sputum but, in younger children who do not expectorate or who are at an early stage of the disease progression, diagnosis rests largely on the history. The only reliable way to make the diagnosis unambiguously is to undertake bronchoscopic examination, but the number of patients is such that this is not currently our first line investigation, particularly as many will resolve completely with one or two courses of antibiotics. Common things are common, and we do try hard to determine whether asthma is the sole diagnosis or a contributory factor through appropriate trials of treatment.

Fortunately our primary care physicians are very good and it is uncommon for a child to be referred who has not been assessed as possibly having asthma. The other paper which focuses on the diagnosis of chronic bronchitis highlights the problem.

We write with regard to the retrospective chart review by Donnelly and colleagues published recently in Thorax. The review covers a 5 year period and it is presented as a cross-sectional survey of their personal practice of children with persistent bacterial bronchitis. We are unsure how long it would take to go over the time of the review and it does not examine outcomes longitudinally.

We agree that there is a lack of clarity regarding the definition of chronic bronchitis. However, like all diagnoses of exclusion, it is a difficult definition. The definition of chronic bronchitis in adults is more specific: “the presence of chronic productive cough for 3 months in each of two successive years and a patient under whom other causes of chronic cough have been excluded”.

Concerning the diagnosis from two recently published paediatric respiratory text books are illuminating. In the first the adult definition of chronic bronchitis is given but “whether this definition can be applied to childhood chronic bronchitis remains unclear” and “it has the potential to defeat the paediatrician from detecting a more specific respiratory condition”. The authors of the chapter suggest that: “The diagnosis of chronic bronchitis should occur in two phases. The first is consideration and identification of several well defined respiratory conditions according to a standard management protocol. The second but simultaneous phase is elimination or modification of exogenous factors that produce or maintain the child’s illness.” The second paediatric text also notes that “the definition of chronic bronchitis in children is less clear ...”.

Our complaints about this paper, based on our own experience, is that the label of “chronic bronchitis” is given to children without adequate exclusion of other diagnoses. We acknowledge that our centres may differ because of the referral pattern, with most of the patients referred from hospital or community paediatricians rather than primary care physicians recognise the potential size of this problem.

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References


Outcomes in children treated for persistent bacterial bronchitis

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persistent endobronchial bacterial infection

adopted the terms secondary phenomenon resulting from children but this, as in the children, is a recurrent/persistent bacterial endobronchial pulmonary disease (COPD) are plagued by Many adult patients with chronic obstructive bronchitis... with appropriate treatment. In a significant proportion it probably leads to bronchiectasis, in a significant proportion of ex-smokers is due to impaired clearance of the airways. This may be due to cystic fibrosis or an immunodeficiency but, most commonly, is secondary to a “hit and run” infect such as a significant viral lower respiratory tract infection or—much less commonly these days—pertussis. Other causes of impaired clearance such as mucus plugging in asthma, tracheomalacia or even pulmonary vascular congestion with congenital heart disease may allow Haemophilus influenzae in particular to colonise the lower airways. Finally, we would wish to clarify some of the misconceptions in the letter by Drs Byrnes and Edwards. We did not say that bronchiectasis frequently resolves in those with immunodeficiency but mention that it has been reported. Patients did not take up to six courses of prolonged antibiotics to improve. This was the time taken to affect a cure and a few will take longer. As previously noted, in the vast majority of cases the cough remains even after high-dose antibiotic failure to show a dramatic response calls the diagnosis into question. However, a small minority may take longer and occasionally do not clear even with 2 weeks of intravenous antibiotics, but have subsequently cleared with nebulised colistin which is active against Haemophilus. Our approach is based on the belief that the lack of a cough suggests that there is no active infection and that, under these conditions, the airways are healing themselves. When the typical cough returns, we aim to treat it aggressively and early until the condition resolves. We believe the main focus of research should be in how to identify the condition early in order to prevent the need for long and, in some cases, recurrent courses of antibiotics once the infection has been present for months or years.

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Competition of interests: None.

References


Authors’ reply

We thank Drs Byrnes and Edwards for their comments regarding our paper1 and would certainly agree that the diagnosis of persistent bacterial bronchitis/persistent bacterial endobronchial infection can be difficult to make. However, without recognition that the condition exists, the diagnosis cannot be made. In our earlier letter (see page 921) we highlight the difficulties in reaching a correct diagnosis in a child with a chronic cough due to the limited repertoire of responses shown by the lungs when inflamed. Failure to identify children with persistent endobronchial infection results in a huge burden of unnecessary and inappropriate treatment. In a significant proportion it probably leads to bronchiectasis, although this may take decades. Our experience is that persistent endobronchial infection in children is curable, irrespective of the CT appearance, providing there is not a significant ongoing underlying problem such as cystic fibrosis or severe immunodeficiency. Bronchiectasis is not a diagnosis; rather, it is a result of a pathological finding at one end of the spectrum from normality through minor peribronchial wall thickening and patchy non-specific changes. We have major concerns regarding the use of CT scans by those who do not understand the natural history of the disease. We have seen a number of patients who were noted to have significant quantities of secretions on bronchoscopy and a heavy growth of one or two organisms in the lavage fluid who were then largely left untreated because the CT scan did not show bronchiectasis.

The letter from Drs Byrnes and Edwards highlights the added problem using a non-specific term such as “chronic bronchiitis”. This is why we and Anne Chang’s group have deliberately adopted the terms “persistent bacterial bronchitis/persistent bacterial endobronchial infection” which highlight the fact that this is persistent endobronchial bacterial infection and is quite distinct from adult “chronic bronchiitis” associated with cigarette smoke. Many adult patients with chronic obstructive pulmonary disease (COPD) are plagued by recurrent/persistent bacterial endobronchial infection whereas in children we see in children but this, as in the children, is a secondary phenomenon resulting from impaired mucociliary clearance. These patients have two ongoing pathologies—one (COPD) predisposing to acquisition of the second (persistent endobronchial bacterial infection). We would go further and speculate that the continuing symptoms and decline in lung function in a significant proportion of ex-smokers is due to ongoing inflammation secondary to persistent endobronchial bacterial infection. As noted above, we believe that persistent endobronchial bacterial infection is not a primary diagnosis but represents colisation secondary to impaired clearance of the airways. This may be due to cystic fibrosis or an immunodeficiency but, most commonly, is secondary to a “hit and run” infect such as a significant viral lower respiratory tract infection or—much less commonly these days—pertussis. Other causes of impaired clearance such as mucus plugging in asthma, tracheomalacia or even pulmonary vascular congestion with congenital heart disease may allow Haemophilus influenzae in particular to colonise the lower airways.

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References


HIV-related TB and adverse drug events

Breen and coworkers1 showed that, in the era of effective antiretroviral therapy, discontinuation of anti-tuberculosis (TB) treatment occurred with a similar frequency in HIV-infected and HIV-uninfected individuals despite a greater rate of serious (grade III/IV) adverse events among HIV-infected individuals. According to the Division of AIDS table for grading the severity of adult and pediatric adverse events (http://rrc.tech-res-intl.com), grade III adverse events are likely to cause inability to perform usual social and functional activities while grade IV adverse events are potentially life-threatening. However, among HIV-infected patients with grade III/IV adverse events in the above study, treatment was interrupted only in a minority of patients, except for those with hepatotoxicity, and no mention was made regarding modification of treatment regimens. With the retrospective study design, it might be difficult to exclude some degree of subjective bias in symptom reporting/grading/interpretation, especially among HIV-infected individuals, despite the use of a standardised grading scheme.

In contrast with previous studies,1-3 anti-TB drug-related hepatotoxicity was observed at a similar rate in HIV-infected and HIV-negative patients. Differing abilities to control socio-demographic and clinical profounders—such as malnutrition, alcohol use, drug abuse, hepatitis B/C, anti-retroviral drugs—could account for the difference, especially with the limited sample sizes of these studies.1 In this regard, it is interesting to note that use of rifampin plus pyrazinamide in the treatment of latent TB infection was associated with apparently higher prevalences of hepatotoxicity in clinical trials conducted among HIV-negative subjects1 than those conducted among HIV-infected individuals. As hepatotoxicity is a major factor leading to interruption of anti-TB treatment, the similar incidence of hepatotoxicity in HIV-infected and HIV-negative patients is perhaps reassuring.

However, while the attending clinicians might be unwilling to interrupt the anti-TB treatment among HIV-infected subjects even in the face of severe vomiting and peripheral neuropathy, patient cooperation could be jeopardised and drug adherence would then be difficult to ensure outside the setting of directly observed therapy. Non-adherence, frequent regimen modifications and treatment interruptions certainly increase the risk of treatment failure and relapse with acquired resistances. With the recent report of highly fatal cases of drug resistant TB among HIV-infected patients, there remains a need for heightened awareness of possible adverse drug events, as well as vigilance in the prevention, detection and management of such events.

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Catherine Byrnes and Elizabeth Edwards

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