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The significance of early-life prevention of COPD in sub-Saharan Africa: findings from the FRESH AIR UGANDA survey

Recent advances in the management of pre-school wheeze

Childhood pneumonia – looking beyond mortality

An unusual case of an anterior mediastinal mass in a child with cystic lung disease

The worst is yet to come: New virus even deadlier than Ebola, Zika may emerge, warn Swiss scientists

In 2014, the Ebola virus outbreak killed more than 11000 people in Africa. This year, the Zika virus is currently affecting millions of people and even unborn children in South America, prompting the declaration of a public health emergency by the World Health Organization (WHO).

In a study published last week in the journal *Proceedings of the National Academy of Sciences*, Swiss scientists warned that a deadlier virus may emerge, causing more illnesses and deaths.

According to a report on WND.com, the Swiss scientists described the virus as something similar to the one which causes severe acute respiratory syndrome (SARS), which killed over 700 people during an outbreak in southern China from November 2002 to July 2003.

The virus, which is called 'WIV1-CoV', may come from zoonotic sources, meaning it may be transmitted from animals to human beings. It is likely to exhibit flu-like symptoms which will eventually escalate into pneumonia.

'Focusing on the SARS-like viruses, the results indicate that the WIV1-coronavirus (CoV) cluster has the ability to directly infect and may undergo limited transmission in human populations,' the researchers wrote in their study.

Lead researcher Dr. Vineet Menachery of Department of Epidemiology, University of North Carolina at Chapel Hill explained that the transmission of this new virus to humans is not yet a certainty, but if it happens, the scenario is discouraging.

'This virus may never jump to humans, but if it does, WIV1-CoV has the potential to seed a new outbreak with significant consequences for both public health and the global economy,' the lead scientist explained.

Menachery added that the capacity of the WIV1-CoV to jump from animals such as bats to human is 'greater' than originally thought.

'While other adaptations may be required to produce an epidemic, several viral strains circulating in bat populations have already overcome the barrier of replication of human cells and suggest re-emergence as a distinct possibility,' the researcher said.

Non-travel-associated Middle East Respiratory Syndrome in Kenya

Scientists studying the zoonotic disease caused by the Middle East respiratory syndrome coronavirus (MERS-CoV) have focused largely on camels, one confirmed animal reservoir of the pathogen. Researchers have also found evidence to suggest the virus may have originated in bats. But a recent finding — MERS antibodies in two people who handle non-camel livestock in northeast Africa — reinforces the idea that camels and bats may not be the only animal reservoirs of MERS-CoV, and suggests that the virus may be endemic to another region, beyond its namesake.

Researchers at the Kenya-based International Livestock Research Institute (ILRI) and at the University of

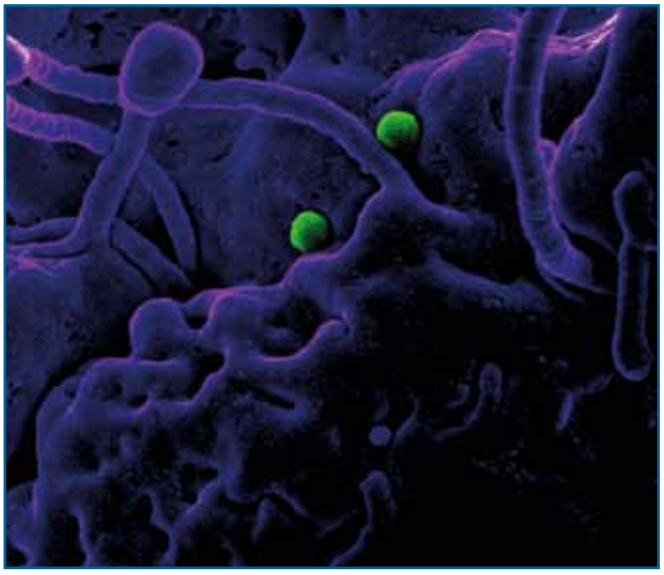


Photo credit: NIAID in collaboration with Colorado State University (MERS Coronavirus Particles) [CC BY 2.0 (http://creativecommons.org/licenses/by/2.0)], via Wikimedia Commons

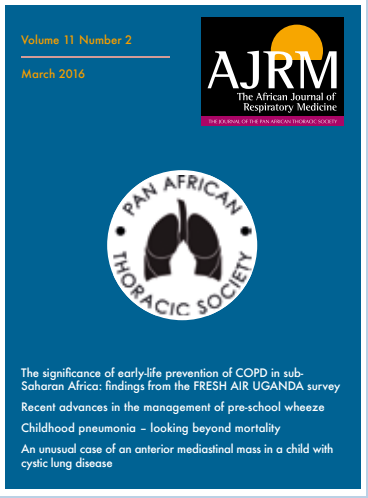
Bonn Medical Centre in Germany have found evidence of MERS-CoV antibodies in archived blood samples from two of 1122 Kenyan livestock handlers, collected between 2013 and 2014. Both people whose blood showed signs of past MERS-CoV infection — a 26-year-old woman and 58-year-old man — were asymptomatic and kept livestock other than camels, the researchers report in May 2016's issue of *Emerging Infectious Diseases* (http://wwwnc.cdc.gov/eid/article/22/6/16-0064_article?platform=hootsuite).

While there have been confirmed cases of MERS outside the Middle East, all of these to date have been linked to people who had travelled to the affected area. This study is the first to report non-travel-associated cases of MERS-CoV infection in Africa.

'The absence of autochthonous human MERS-CoV infections in Africa has triggered hypotheses regarding differences in disease transmission between Africa and the Arabian Peninsula, and has raised doubts regarding the role of camels as a source of infection,' the researchers wrote in their paper. 'Our study provides evidence for unrecorded human MERS-CoV infections in Kenya and the proportion of seropositive specimens that we found is comparable to previously reported proportions of unrecorded infections in the general population in Saudi Arabia.'

The finding provides additional clues for epidemiologists studying the origins and spread of MERS. 'The presence of antibodies in two people in Kenya suggests that the disease may have been endemic in the area since time immemorial,' said Kenneth Mbai, a lecturer in veterinary medicine at University of Nairobi, who was not involved in the research.

Thomas Manga, an assistant director of veterinary services in Kenya's livestock development ministry, said additional evidence for this immunity hypothesis is needed. 'The presence of antibodies to the virus indicates that the people were infected with the virus sometime in the past and... is not necessarily associated with clinical disease,' said Manga, who was not involved in the research.



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First word

It is my pleasure and privilege to extend an invitation to all healthcare workers involved in lung health throughout Africa and the world to the 1st PATS International Conference. The conference is going to take place in Nairobi, Kenya from 9th April to 12th April 2016.

The Pan African Thoracic Society was formed in 2003 to create a representative African respiratory society for the region to address the high burden of respiratory illness and to promote lung health in Africa through education, training, research, and advocacy.

This is the first conference of the PATS since its inception. The conference will bring together a gathering of healthcare workers from across Africa, including physicians, paediatricians, general practitioners, thoracic surgeons, and other health workers involved in lung health in the continent.

The theme of the conference, 'Breathing life into Africa', will give participants an opportunity to focus attention on the most pressing issues for lung health. The congress will provide a platform for the presentation of up-to-date reviews, as well as dissemination and discussion of results of cutting-edge research findings that will have wide implications for the practice of evidence-based respiratory care.

In addition, the congress will provide an excellent opportunity for collaboration and brainstorming on productive advocacy pathways for tackling the challenge of respiratory diseases in Africa. It will also provide an opportunity to strengthen the role of PATS in promoting respiratory healthcare in the continent.

The faculty will include global and African leaders in lung health, as well as key representatives from leading international societies involved in lung health across the globe, including the European Respiratory Society (ERS), American Respiratory Society (ARS), South African Respiratory Society (SARS), International Union Against Tuberculosis and Lung Diseases (IUATLD), and International Forum of Respiratory Societies (IFRS). To ensure meaningful participation and assured outcomes, representatives of these diverse bodies who are participating in the congress have been actively engaged in its planning and organisation.

Last but not least, the first PATS conference will provide an effective opportunity for meeting friends and colleagues who are engaged in respiratory heath in Africa and globally.

On behalf of the PATS 2016 conference Organising Committee, I would like to encourage and welcome you to take part, and benefit from attending this major respiratory conference.

More information can be found on the website www.pats.2016.com

Dr Joseph A. Aluoch, FRCP, EBS, Chairman, Organising Committee, Pan African Thoracic Society 2016 Conference

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The Pan African Thoracic Society exists to promote respiratory health in Africa. It is supported by the Nuffield Foundation (UK) and the American Thoracic Society.
www.africanthoracic.org

The significance of early-life prevention of COPD in sub-Saharan Africa: findings from the FRESH AIR UGANDA survey

FV Gemert, B Kirenga, R Jones

Abstract

Chronic obstructive pulmonary disease (COPD) is globally one of the major non-communicable diseases (NCDs). COPD is also among the NCDs that are increasing worldwide, with the poorest and most vulnerable communities most affected. For decades, tobacco smoke (including second-hand smoke or passive exposure) has traditionally been seen as the primary cause of COPD. In Africa and other low-income settings, biomass exposure is probably the most important risk factor for COPD. In Western communities, where COPD is mainly due to tobacco smoking, the disease becomes clinically apparent particularly in the fourth and fifth decade of life because the pathological events that lead to COPD develop slowly over a long period of time. It is plausible that where biomass smoke exposure starts early in life, even prenatally, that COPD can develop much earlier. The prevalence of COPD in people younger than 40 years is not known because most surveys have included people 40 years or older. The FRESH AIR survey included people above the age of 30 years. Results show that the prevalence of COPD among adults aged 30–39 years was 39% (38% in men and 40% in women). COPD surveys among young people in sub-Saharan Africa are urgently needed. Education programmes to raise awareness and knowledge of the risk of COPD in general, and early-life COPD in particular, are needed.

Introduction

Chronic obstructive pulmonary disease (COPD) is globally one of the major non-communicable diseases (NCDs), and an important contributor to the global burden of disease in people older than 40 years.[1] COPD is also among the NCDs that are increasing worldwide, with the poorest and most vulnerable communities most affected.[2,3] In 2010, COPD was the fourth highest cause of death globally and it was expected to become the third by 2030. In 2014, the World Health Organization (WHO) reported that COPD is now the third leading cause of mortality, and has surpassed the combined mortality of tuberculosis, HIV/AIDS and malaria in sub-Saharan Africa.[4]

For decades, tobacco smoke (including second-hand smoke or passive exposure) has traditionally been seen as the primary cause of COPD, affecting more than 200 million people worldwide. These estimates, however, were based on data from studies conducted in industrialised countries.[5] The major prevalence studies were particularly conducted among participants above the age of 40 years living in urban areas.[6–8]

It is now recognised that a substantial proportion of COPD cases (up to 20%) can not be explained by exposure to tobacco smoke, particularly in low- and middle-income countries (LMICs). During the last decade, other risk factors have been found to be associated with the development of COPD, such as indoor and outdoor pollution, environmental exposures, untreated asthma and tuberculosis, as well as dietary and genetic factors.[9–12] In Western communities, where COPD is mainly due to tobacco smoking, the disease becomes clinically apparent especially in the fourth and fifth decade of life. This is because the pathological processes that lead to COPD are slow and accumulate over time. In biomass smoke-related COPD, exposure starts much earlier in life, and often even in prenatally. This early exposure means that long durations of exposure can be attained in early life, hence COPD caused by biomass smoke exposure can occur early in life. In addition, growing attention is being given to the fact that early life disadvantages could predispose the young individuals to a higher risk of developing COPD in adult life as well.[10–13] The FRESH AIR Uganda survey is one of the very few COPD surveys that has specifically included people younger than 40 years. It is hoped that these findings will stimulate research into COPD among the young, allowing design of preventive interventions in early life.

FRESH AIR Uganda

In 2012, a prospective cross-sectional survey (FRESH AIR Uganda) was performed among a representative sample of 588 participants above the age of 30 years on the prevalence of COPD and its risk factors in a rural district of Masindi in Uganda.[14] Pre- and post-bronchodilator spirometry was per-

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formed by well-trained local healthcare workers working at the different health centres of Masindi district. The survey used the lower limit of normal threshold, i.e. participants below the fifth percentile of the predicted FEV_1/FVC ratio (forced expiratory volume in one second/forced vital capacity) as the defining criterion of COPD instead of the fixed ratio $FEV_1/FVC<0.7$, to avoid under-diagnosis in young participants and over-diagnosis in elderly participants.[15,16]

Among adults above the age of 30 years, we found a prevalence of spirometry-based COPD of 16% (53% women). The prevalence was unexpectedly high among adults aged 30–39 years: 39% (38% men and 40% women). Among the participants with COPD, 44% of the men and 8% of the women were current smokers; their mean age was 40 and 52 years respectively (in the group aged 30–39 years, 65% of men and 5% of women were current smokers). In addition to tobacco smoking, almost 95% of the participants were exposed to smoke caused by biomass fuel use.[14]

Discussion

The findings of this survey indicate that a high proportion of COPD patients are younger than the universally accepted 40 years. Why would COPD develop in young people in Sub-Saharan Africa? What should be done?

Indoor air pollution

While the development of COPD is multifactorial, indoor air pollution arising from biomass fuel use is probably the most important risk for COPD in LMICs.[17] Around three billion people, most of them living in LMICs, rely on the use of open fires and simple burning of biomass fuels (wood, animal dung, crop residues, straw and charcoal) for cooking and heating. The smoke produces high levels of household air pollution with a range of health-damaging pollutants. These include significant amounts of particulate matter, of which the smallest particles (mean aerodynamic diameter of particles <2.5 μg, PM2.5), can penetrate deep into the lungs to the alveolar spaces.[11,12] Household air pollution often affects the poorest households that are unable to afford clean, efficient cooking practices. In most countries of sub-Saharan Africa, women have the responsibility for domestic cooking, and most are exposed to biomass smoke each day, as well as the young children and the sick who spend more time indoors or around cooking fires.

Early life events and impact on lung growth

A second explanation for the high prevalence of COPD in sub-Saharan countries may be related to early-life events. In normal lung development, airway branching is complete by the first trimester of pregnancy.[18] Alveoli develop by a different process and are present at birth, increasing during childhood.[13] Lung volume and airflow continue to increase as the thorax grows; lung growth ceases in young adulthood (by the end of adolescence in women and by the mid-20s in men) and lung function remains constant for about 10 years (plateau phase); thereafter lung function slowly decreases.13–19 It is already known that early-life events leading to low birth weight, increase the risk of developing COPD.[20]

When a pregnant woman is cooking, the exposure to biomass smoke affects her baby: this is associated with low birthweight, and reduced lung function of the infant soon after birth.[21] Thereafter, young children are exposed to these high levels in the first years of their lives, as they remain close by their mother during cooking (infants often carried on the back of their mother), causing poor lung growth and reduced development of the lungs during childhood.[11,12] Biomass smoke also induces respiratory infections among young children, strongly associated with a decline in lung function in later life.[22] Exposure to household air pollution accounts for more than half of deaths to childhood pneumonia in children under five years of age.[12] The reduced lung function due to biomass smoke exposure among children continues during life, and could result in a lower plateau at young adulthood, having a lasting effect into adulthood, and thus substantially increasing COPD risk.[19]

Facing the variety of COPD risk factors in sub-Saharan Africa

A person living in a rural area of sub-Saharan Africa has lifelong exposure to a variety of risk factors for the development of COPD during all stages of life: perinatal factors (maternal exposure to biomass smoke, low-birth weight, and pre-term birth), childhood exposure (respiratory tract infections, exposure to indoor biomass smoke, childhood asthma, second-hand smoking, occupational exposures, poor nutrition, and kerosene lamps), and adult exposure (occupational exposures, agricultural smoke, indoor biomass smoke, cigarette smoking, second-hand smoking, kerosene lamps, and outdoor air pollution). Outdoor air pollution is a growing problem, especially in cities.[23,24] Tobacco smoke also exacerbates the detrimental effect of biomass smoke.[17,25] There is an inverse relationship between socioeconomic status and male smoking prevalence.[26] Socioeconomic indicators such as poverty are also associated with poor access to healthcare, poor nutrition, low birth weight, exposure to indoor and outdoor air pollution, poor living conditions and water supply/sanitation, causing ill health effects, and therefore increasing the risk of developing COPD.[26–29] The influence of socioeconomic factors is complex: more research is needed to identify these partly modifiable, and at the same time independent, risk factors.[27,28,30]

Educational programmes

Most people (communities, healthcare professionals, and policy-makers) are unaware of the damage caused by biomass smoke. Lung health education programmes offer the potential to teach people about the problem of biomass smoke exposure and allow those at greatest risk, including pregnant women and young children, to change their cooking traditions and apply behavioral changes to the exposure of household air pollution. A cascading and sustainable 'train-the-trainers' module, such as is being run by the FRESH AIR Global Bridges project in the Masindi area of Uganda, offers an example of how this education may be carried out. Educational materials developed by local health workers have now been approved by the Ministry of Health in Uganda.[1,2]

Conclusion

A silent growing epidemic of COPD, starting at younger ages,

seems to be developing in sub-Saharan Africa. This applies particularly to communities living in rural areas, an often neglected group of people. Although tobacco smoking remains an important cause of COPD, almost all are exposed to biomass smoke and this interacts with other risk factors such as early-life respiratory infections, tuberculosis, low birth weight, poverty, and malnutrition. Although prevention of COPD can be achieved to some extent by smoking cessation, reduction of the exposure to the other risk factors should be a major public health goal for LMICs, and should start early in life. Public awareness and control of the household environment are important steps in preventing respiratory diseases. While research on the effects of biomass smoke is increasing, additional research is needed into the benefits of prevention and reductions of exposures at a community level. Researchers, policy-makers and government, stakeholders, health professionals, and communities will have to work together to control the growing burden of COPD, and start prevention and intervention programmes.

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Recent advances in the management of pre-school wheeze

A Bush

Abstract

Pre-school wheeze is very common and often difficult to treat. Most children do not require any investigations, only a detailed history and physical examination to ensure an alternative diagnosis is not being missed; the differential diagnosis, and hence investigation protocols for the child in whom a major illness is suspected, shows geographical variation. The pattern of symptoms should be divided into episodic viral and multiple trigger to guide treatment, and the pattern of symptoms re-assessed regularly. Attention to the proper use of spacers, and adverse environmental exposures such as tobacco smoke exposure, is essential. There are no disease-modifying therapies, so therapy is symptomatic. This paper reviews recent advances in treatment, specifically new data on the place of leukotriene receptor antagonists, prednisolone for acute attacks of wheeze and antibiotics, and proposes treatment protocols for the two types of wheeze.

Introduction

In some parts of the world, more than 30% of infants are reported to wheeze before age three years.[1] and many are very difficult therapeutic problems. This paper proposes a framework for management of these infants, based on modern insights into pathophysiology. It should be stressed it is written from a European perspective; the reader should evaluate critically the extent to which it applies to Africa, especially in a low- and middle-income setting.

Initial approach: am I missing a diagnosis?

Especially in the very young, specific diagnoses should be considered; there will be marked geographical variation in the differential, for example airway compression by tuberculous lymph nodes is common in parts of Africa, but very rare in the United Kingdom. The first step is as always to take a good history and perform a detailed physical examination. This should determine whether the sound heard is really wheezing (I am always sceptical until a reliable paediatrician has actually heard a wheeze), whether they get breathless, and if in fact what was complained of is an isolated dry cough, which in a community context in a well child is unlikely to betoken significant disease.[2] The pattern of symptoms should be determined because this will determine treatment (below). Red flags that more detailed assessments are needed are given in Table 1.

Coughs and wheezes can be divided into five categories (Table 2).[3] In my practice, 'Nursery School syndrome' is commonest. This afflicts children placed early into a child care facility, often first-time parents; the child gets a succession of viral colds (ten/year, with two weeks of symptoms with each cold being well within the normal range) with very few healthy days in between each cold. Of course, symptoms do not respond to inhalers or antibiotics; reassurance is what is needed. The most important lesson is that, before abnormality can be diagnosed, the paediatrician must be fully familiar with the limits of normality.[4]

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Planning treatment: how?

If on the basis of history and examination it is decided to treat the child for an asthma syndrome, the first step is to determine if the child only has symptoms at the time of viral colds (episodic viral wheeze, EVW) or in addition has symptoms between viral colds triggered by, for example, excited behaviour, allergen exposure (multiple trigger wheeze, MTW).[5] These clinical phenotypes may change over time, and detailed re-assessment at regular intervals is essential. This distinction is not merely of academic interest; preschool children with MTW, but not EVW have eosinophilic airway inflammation,[6] and this has implications for treatment: the use of inhaled corticosteroids (ICS) is only likely to be successful in eosinophilic airway disease, a point which is discussed further below. The many brilliant epidemiological studies that classify wheeze phenotypes can only be applied retrospectively, and currently are not useful in planning treatment.[1,7,8]

In theory, treatment of the pre-school child with wheeze could aim to prevent the transition to established asthma, or treat symptoms. We have no medications that can prevent progression to asthma; three excellent studies have shown that ICS given early as preventive therapy do not work,[9–11] and the pathological correlate of this is the complete absence of inflammation in very early wheeze.[12] Hence symptomatic treatment is appropriate, including intermittent therapy for intermittent symptoms.

Treatment options: what?

Before escalating pharmacotherapy, it is important to ensure the environment is optimal, especially that tobacco smoke exposure is eliminated, and any inhaled medications

Red flags on the history	Red flags on the physical examination
Prominent upper airway symptoms Symptoms from first day of life Sudden-onset symptoms, which always suggests a foreign body Chronic moist cough/sputum >8 weeks duration every day Worse after meals, irritable feeder, arches back, vomits, suggests gastro-oesophageal reflux Systemic illness or immunodeficiency Continuous, unremitting symptoms	Clubbing, weight loss, failure to thrive Upper airway disease – tonsillar hypertrophy, rhinitis, nasal polyps which last mandates consideration of cystic fibrosis Unusually severe chest deformity Fixed monophonic wheeze, stridor, asymmetrical signs Signs of cardiac or systemic disease

Table 1: Red flags on history and physical examination, which should prompt consideration of more detailed investigations

Diagnostic category	Examples
Normal child (the hardest diagnosis!)	Recurrent viral colds Pertussis
Serious illness	Will show regional variation; likely includes TB and bronchiectasis in Africa
An 'asthma syndrome'	Episodic viral wheeze Multiple trigger wheeze
Minor mimics or exacerbators of symptoms	Allergic or infective rhinitis Gastro-oesophageal reflux
Over-anxious parents	Often first-time parents who do not appreciate the range of normality Find out if they have some concealed fear, e.g. a friend's child died of TB having had a non-specific presentation

Table 2: A pre-school child with cough or wheeze will fall into one of these five categories

are properly administered. It is also important to consider whether treatment is needed at all; if the child merely has noisy breathing but remains well otherwise, then doing nothing is almost certainly appropriate. The simplest therapies are intermittent bronchodilators – either anticholinergics or short-acting β-2 agonists – via a mask and spacer. There is no way of predicting responses in an individual child, and a therapeutic trial is indicated. The next series of options are oral leukotriene receptor antagonists, ICS, and, controversially, antibiotics. Each will be considered in turn.

Leukotriene receptor antagonists

Respiratory viral infections have long been known to be associated with an elevation in cysteinyl leukotrienes,[13] and intermittent and continuous montelukast has been suggested as a treatment strategy. However, recent trials[14–17] have not been encouraging (Table 3). In summary, the two largest recent trials, recruiting over 3000 children, have failed to show benefit for montelukast. Hence although anecdotally a few individuals may respond to montelukast, most will not. There is no way we can determine which rare individuals will respond, except by a therapeutic trial. It should be noted that the behavioural side-effects of montelukast are not trivial. In summary, for the vast majority of pre-school wheezers, therapy with montelukast has no place.

ICS

Relevant studies using ICS are summarised in Table 4.[15,18–20] The very high dose intermittent ICS regime showed benefit, but at a cost of growth suppression;[10] and considering how many viral colds a child may have, this high dose cannot be recommended. We know that continuous inhaled or nebulised steroids are ineffective in preventing EVW. If the attacks are really so severe that it is felt that something must be done then a trial of ICS for a defined and well-monitored period (Dutch regime) may be indicated,[21] but they should be discontinued if there is no benefit (the likely scenario). There is limited evidence for acute benefit of ICS, and the risks are not small if high doses are used; I would not use doses above 200 µg beclomethasone equivalent as acute intermittent therapy. Atopy is not helpful in predicting ICS response in preschool wheeze.

The A-word: what is the role of antibiotics?

The role of bacteria in exacerbations of airway disease has increasingly come to prominence. In a study of adults with viral colds, co-amoxyclav significantly shortened the duration of symptoms, but only in those with a positive upper airway bacterial culture.[22] In a recent study, bacteria and viruses were equally likely to be cultured from the upper airway.[23] However, the mere presence of bacteria does not mean they are of pathophysiological significance; it might merely be that viral infection causes a transient local immune paresis leading to secondary bacterial colonization. A recent highly controversial study from Denmark[24] randomised 72 children aged 1–3 years with 158 asthma-like episodes lasting at least three days to azithromycin or placebo for 3 days. Symptoms were shortened, especially if azithromycin was started early (less than six days after the onset of symptoms). For the majority of children, no

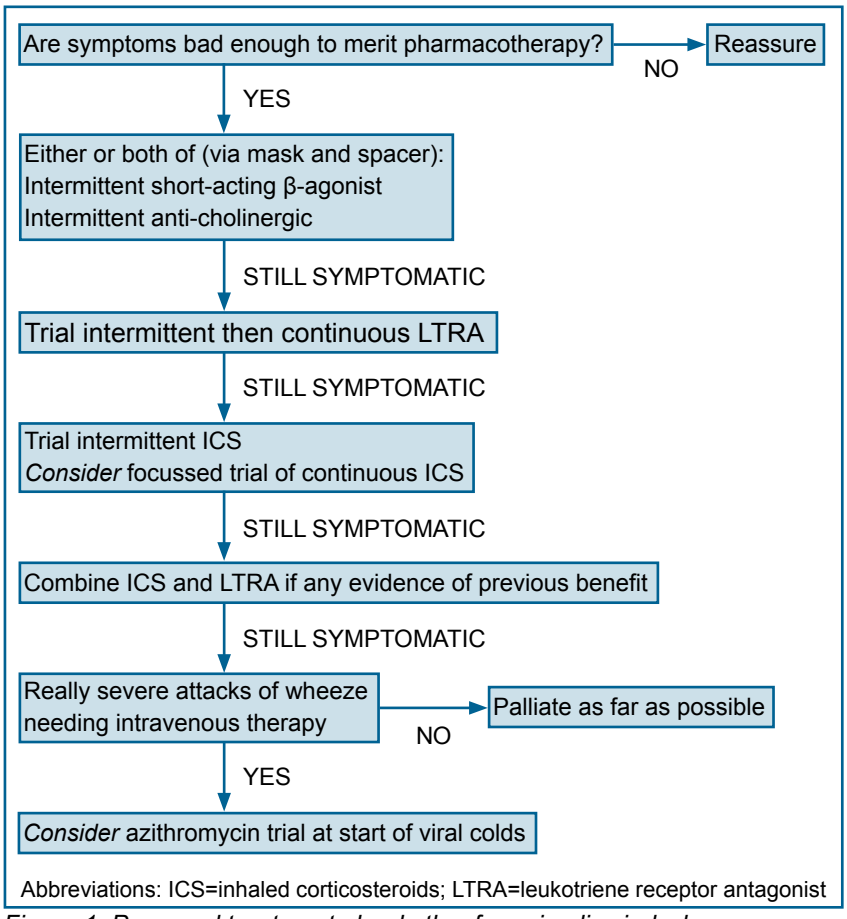

Figure 1: Proposed treatment algorhythm for episodic viral wheeze.

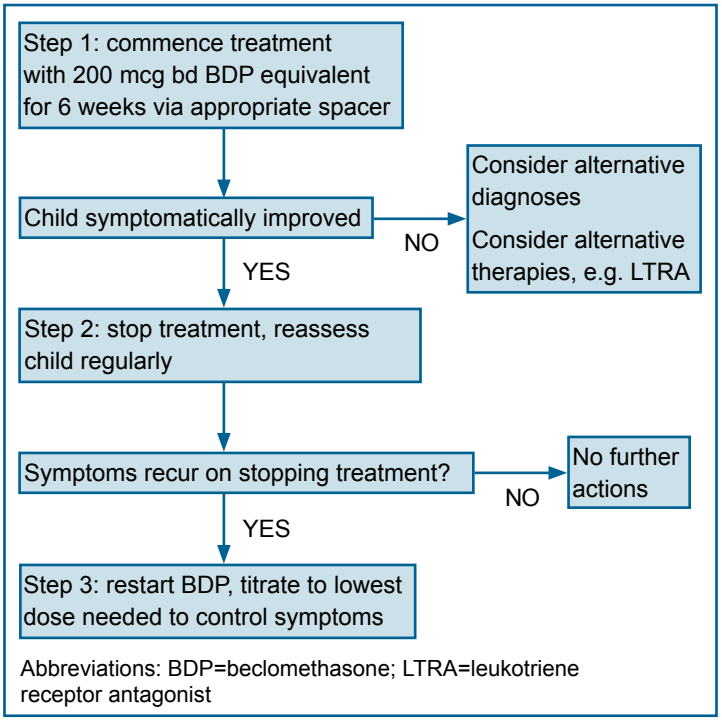

Figure 2: Proposed treatment algorhythm for multiple trigger wheeze.

bacterial cultures results were reported. It is of course unclear whether the effects of azithromycin were mediated by antibacterial or any of the many different immunological effects of the medication.[25] This could have been resolved if there had been a third limb to the trial, using (for example) co-amoxyclav.

What then is the role of azithromycin in pre-school wheeze? Clearly if every child with a cold is prescribed azithromycin, azithromycin resistance in the community will rise dramatically and azithromycin will cease to be useful. There can be no justification for the routine prescription of antibiotics to children with viral colds, irrespective of whether a positive bacterial upper airway culture is found. Azithromycin can only be justified as a trial in pre-school children with wheeze so severe that they require at least intravenous treatment and oxygen, and should only be continued if it prevents hospital admission.

Prednisolone in pre-school wheeze

Two recent studies[26,27] involving nearly 1000 children have clarified the role of prednisolone in pre-school children. It is clear that if the child is well enough to be looked after in the community, then prednisolone does not need to be prescribed. Furthermore, most children admitted to hospital will not need prednisolone. Oral steroids are only needed in really severe pre-school wheeze, for example if the child is oxygen dependent and intravenous treatment is being contemplated.

Treatment protocols: EVW and MTW

EVW

A proposed treatment flow chart is given in Figure 1. The evidence base is scanty and the diagram reflects personal practice. In all cases, if a treatment approach is not working, it should be discontinued without hesitation.

MTW

Preventive treatment is recommended in the following situations: if the child has symptoms which respond to short-acting β-2 agonists at least three days a week in between viral colds; if attacks of viral wheeze are very severe (although this is not likely to be a good strategy, and should certainly be discontinued if there is no evidence of benefit); and if symptom under-reporting by the parents is suspected. A three-stage flow chart is given in Figure 2. It is essential to have a trial of stopping an apparently successful treatment, because many children improve spontaneously, and without this step, unnecessary treatment will be prolonged

Summary and conclusions

The vast majority of pre-school children who wheeze do not need any investigations, but only a careful history and physical examination to ensure there are no features

suspicious of an alternative diagnosis. The key is to be sure that the noises the family describe are really wheeze, and keep an open mind until a physician has actually heard the noises. For the purposes of treatment, classify pre-school wheeze as 'episodic (viral)' and 'multi-trigger', but keep re-assessing the child, because these phenotypes may change over time, and hence treatment may need to change. Since there are no disease-modifying therapies, treatment is symptomatic, and episodic symptoms can be treated episodically, assuming they are severe enough to merit treatment. If preventive therapy is being trialled, a three-step protocol is mandatory to avoid over-treating the child. There is no justification for the routine use of oral antibiotics with viral colds, despite recent data. Finally, oral corticosteroids have been over-prescribed in the past for acute attacks of pre-school wheeze and should be reserved for very severe attacks.

Reference	Intervention	Numbers	Result
Robertson et al[14]	Intermittent ML vs placebo	220	Intermittent ML superior to placebo
Bacharier et al[15]	Intermittent ML vs intermittent nebulised BUD vs placebo	238	Intermittent ML and nebulised BUD equivalent and better than placebo
Valovirta et al[16]	Intermittent ML vs continuous ML vs placebo	1771	No benefit of either ML regime
Nwokoro et al[17]	Intermittent ML vs placebo Sub analysis by ALOX5 promoter polymorphisms	1346	No benefit of ML Possible benefit of ALOX5 promoter genotyping
Abbreviations: BUD, budesonide; ML, montelukast			

Table 3: Recent large trials of montelukast in episodic wheeze

Author	Intervention	Numbers	Result
Wilson et al[18]	Regular inhaled BUD 200 µg bd vs placebo	40	No effect of BUD on episodes of wheeze
Bacharier et al[15]	Intermittent ML vs intermittent nebulised BUD vs placebo	238	Intermittent ML and nebulised BUD equivalent and better than placebo
Ducharme et al[19]	Intermittent FP 1.5 mg/day vs placebo	129	Less use of prednisolone in FP group
Zeiger et al[20]	Intermittent nebulised BUD vs continuous nebulised BUD (no placebo)	278	No difference between the regimes
Abbreviations: BUD, budesonide; FP, fluticasone propionate; ICS, inhaled corticosteroids			

Table 4: Relevant studies of ICS in episodic wheeze in pre-school children

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Childhood pneumonia – looking beyond mortality

H J Zar

Introduction

Pneumonia remains a major cause of morbidity and mortality in children globally despite a substantial decline in the incidence in the last decade.[1–3] Advances in immunisation, improvements in socio-economic status, and effective HIV preventative and treatment strategies have reduced the burden of childhood pneumonia and severe disease.[4] However, recent estimates are that globally pneumonia still causes approximately 15% (or just under 1 million deaths) of an estimated 6.3 million deaths annually in children under five years of age.[1] This burden is disproportionately high in Africa, where almost 50% of deaths in children under 5 years occur, despite African children comprising only 25% of live births globally.[1] Of concern, this proportion is estimated to increase by 2030, when African children are projected to account for 50% of global under-five mortality.[1] Pneumonia is the main cause of death in African children outside the neonatal period, accounting for approximately 18% of deaths in children under five years. Among African countries, Nigeria and the Democratic Republic of Congo (DRC) have an especially high burden of childhood pneumonia. Pneumonia incidence and severity is highest in the first year of life, especially in the first six months.[3]

Why is the burden and severity of childhood pneumonia so high in African children and what are the possible interventions to address this?

Five factors will be considered in the following discussion. Firstly, risk factors that may make a child vulnerable to pneumonia or to severe disease may be especially prevalent in some African countries. Paediatric HIV is largely confined to sub-Saharan Africa, where 90% of HIV-infected children reside; HIV infection is an important risk factor for pneumonia, severe disease, and mortality, with HIV-infected children having a six-fold higher risk of developing severe pneumonia compared with uninfected children and a six-fold higher risk of death.[5] Other risk factors for pneumonia and for severe disease that are prevalent in African populations include smoke exposure, lack of breastfeeding, chronic underlying disease, prematurity or low-birth weight, low socio-economic status, crowded living conditions, or malnutrition.[6,7] HIV-exposed but uninfected children, who are born to an HIV-infected mother, are emerging as another important vulnerable group who have a higher risk of pneumonia than HIV-unexposed infants.[8]

Secondly, lack of good access to care and unavailability of effective management strategies is a further factor making African children more at risk of developing severe disease and of mortality. Use of case management guidelines, with effective use of antibiotics has been shown to substantially reduce pneumonia and all-cause under-five mortality.[9] Community-based interventions, including use of case management guidelines by community workers has also been found to effective for reducing pneumonia mortality.[10] Use of oxygen for hypoxic disease is an effective intervention and is life saving.[11] However, almost 50% of African children with pneumonia are not taken to a health facility. Furthermore, the availability and affordability of some essential drugs including oxygen in many African countries is sub-optimal.[12]

Thirdly, there is limited coverage and affordability of effective preventive interventions for childhood pneumonia in Africa. Vaccination is one of the most effective strategies for reducing childhood pneumonia, but coverage for childhood immunisations, especially the newer conjugate vaccines, is also suboptimal in several African countries. New conjugate vaccines against Streptococcus pneumoniae (PCV) and Haemophilus influenzae type b (Hib) have substantially reduced the burden of childhood pneumonia in vaccinated children.[13] Data from six studies of the effectiveness of Hib conjugate vaccine in low- and middle-income countries (LMICs) indicates a reduction of 18% in radiological pneumonia, of 6% in severe pneumonia, and of 7% in pneumonia-associated mortality.[13] While PCV reduces severe invasive pneumococcal disease and bacteraemia, prevention of non-bacteraemic pneumococcal pneumonia is almost 20-fold greater compared with that of bacteraemic pneumonia.[14] Furthermore, the overall burden of disease prevented in HIV-infected children is much greater because of their susceptibility to disease. PCV has also led to a decline in hospitalisation and death for adult pneumonia due to indirect protection through reduction in circulating pneumonia causing pneumococci serotypes.[15] However, a South African study found that even with high coverage for the 13-valent PCV, the incidence of pneumonia remained high, especially in the first six months of life.[8] PCV may also have considerable impact on reducing mortality, as demonstrated by a randomised controlled trial of 9-valent PCV in the Gambia, in which PCV9 reduced childhood mortality by

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15%.[16] Early use of antiretroviral therapy and cotrimoxazole prophylaxis in HIV-infected infants is also effective for reducing pneumonia incidence and severity in children,[17] but coverage for these is still sub-optimal in some areas of sub-Saharan Africa.

Fourthly, African children may have high exposure to infectious diseases compared with children in high-income countries, as well as higher exposure to particular pathogens. For example Mycobacterium tuberculosis (TB) has now been well described to occur commonly in children with acute pneumonia living in high TB prevalence areas, with up to 8% of children presenting with pneumonia found to have culture-confirmed TB.[18] Children living in households in which a member is HIV-infected may be especially exposed to infectious risks and so are more vulnerable to developing pneumonia from a variety of pathogens.

Lastly, the large burden of childhood pneumonia in African children may reflect a young population in which children constitute 25–60% of the total population. However, the burden of pneumonia in African children is disproportionate to the childhood population, with a much higher incidence in African children compared with those in high-income countries.

Discussion

Importantly, the impact of childhood pneumonia extends beyond acute disease in childhood. This is especially relevant as health systems are challenged to address the Sustainable Development Goals for 2030. Chronic sequelae from early childhood pneumonia such as bronchiectasis are increasingly recognised; one review reported chronic sequelae following severe pneumonia to occur in approximately 15% of children.[19] In addition, early childhood pneumonia has increasingly been associated with the development of chronic non-communicable respiratory diseases into childhood and adulthood, such as asthma or chronic obstructive airway disease (COPD).[20–22] Early respiratory syncytial virus (RSV) or rhinovirus-associated lower respiratory tract infection has been associated with the development of asthma in childhood in high-income populations.[23,24] Accumulating evidence from several cohort studies has shown that lung health is established early in life and that lung function follows a set trajectory into adulthood, implying that the roots of adult lung disease such as COPD lie in early exposures including childhood pneumonia.[25]

Effective strategies to prevent and manage pneumonia in African children must be urgently strengthened given the substantial burden of disease, impact on mortality and the accumulating evidence of the association with development of chronic lung disease and respiratory non-communicable diseases (NCDs). The strengthening and implementation of available effective interventions, such as available immunisations and use of case management have the potential to substantially reduce pneumonia burden and under-five mortality.[13] Further strategies to reduce risk factors, such as optimising nutrition, promoting breastfeeding, preventing HIV transmission through mother-to-child prevention programmes, and reducing exposure to biomass or cigarette smoke must be strengthened.

In addition, new strategies are needed to address the residual burden of pneumonia once available vaccines have been well implemented and to develop effective interventions for pathogens for which there are limited strategies. In children well vaccinated with available vaccines including PCV13, viruses can be expected to form an increasing proportion of pneumonia caseload.[26] Among these, RSV is clearly the most prominent pathogen for which there are very limited, affordable interventions available to prevent or treat disease. In 2005, RSV was estimated to cause approximately 34 million episodes of acute lower respiratory tract infection (ALRI) in children under five years or 22% of all ALRI; 10% of episodes resulted in severe illness and hospitalisation and 99% of deaths occurred in LMICs.[27] Furthermore, studies in African children preceding the availability of PCVs[28–30] and several recent case control studies in children well vaccinated with PCV13, mostly from high-income countries, have reported RSV to be a predominant pathogen in children hospitalised with pneumonia.[31,32] Most episodes of severe RSV pneumonia occur in the first few months of life. Therefore the development of several new RSV vaccine candidates is a very promising development. Recently, a novel strategy to immunise pregnant women in the third trimester of pregnancy, enabling transplacental transfer of antibody and potential protection against RSV disease in their infants in the first few months if life has been proposed with the first multicenter trial of RSV vaccination of pregnant women currently underway. This will most likely need to be coupled with additional vaccination of infants to provide extended protection until children are two years of age. The challenge for African countries will be to ensure the availability and affordability of new strategies such as maternal immunisation against RSV, if they are to be effective. Implementation and access to effective vaccines to prevent childhood pneumonia have been much slower in Africa than in high-income countries, but the potential to reduce disease burden is much higher in Africa. For the future, timely access and implementation of such strategies for all children and mothers who need this treatment must be ensured.

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An unusual case of an anterior mediastinal mass in a child with cystic lung disease

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Introduction

A seven-month-old, HIV-unexposed male, of a mixed race ethnicity, presented to a district hospital with a six-day history of progressive shortness of breath and increased work of breathing, associated with a two-day history of fever, vomiting, and pyrexia. He was also noted to have a diffuse vesicular rash thought to be varicella zoster. There was no history of night sweats; however, a poor appetite and weight loss was noted by the mother. The birth history was uneventful and all immunisations were up to date. He was exclusively breastfed for six months with solids introduced at seven months. Development was age appropriate and he was thriving prior to presentation. The chest radiograph showed a pneumothorax on the left (see Figure 1) and an intercostal drain was inserted.

He was transferred to a secondary level hospital where a differential diagnosis of varicella pneumonia or pneumonia secondary to Stahphylococcus aureus with pneumatoceles was made. Examination revealed a hyperpigmented papular vesicular rash with a symmetrical distribution mainly in the trunk, groin, neck, scalp, and both the hands and feet (Figure 2). The chest radiograph showed multiple cystic lesions with a possible mediastinal mass that filled the right hemithorax on the frontal projection. The computed tomography (CT) scan showed a heterogeneous enhancing large anterior mediastinal mass. No calcifications or cysts were noted (Figure 3). The mass extended from the thoracic inlet and was abutting the right hemidiaphgram. No compression of the vessels or airways was noted. There was also no mediastinal lymphadenopathy. Scattered throughout the lungs were multiple small and large walled cysts (Figure 4). The largest cysts were present in the lingula and caused mediastinal shift to the right. The child completed seven days of intravenous cloxacillin and acyclovir and was transferred to a tertiary centre for further diagnostic workup and management.

On arrival at the tertiary centre, he was found to be severely underweight. He was moderately distressed with respiratory rate of 55, ala flaring and subcostal recessions with an oxygen saturation of 95% on one litre of nasal prong oxygen. He had no lymphadenopathy and his skin had a disseminated healing rash with punched out lesions. The chest examination revealed a central trachea with dullness and decreased air entry on the right compared with the left. There was no hepatomegaly or splenomegaly. Investigations showed a raised white cell count with a microcytic anaemia and a normal erythrocyte sedimentation rate (ESR). Beta-human chorionic gonadotropin, alfa-feto protein, uric acid, and lactate dehydrogenase were normal (see Table 1).

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Investigations

Punch biopsies of skin from the left groin and upper back were submitted for histology. Microscopic sections showed fragments of intact skin with spongiotic, hyperkeratotic epidermis demonstrating an infiltrate of Langerhans cells within the superficial dermis (Figure 5). These cells had lobulated, folded vesicular nuclei and abundant eosinophilic cytoplasm. Occasional eosinophils were also seen (Figure 6). The Langerhans cells were positive with the immunohistochemical markers, S100 and CD1a. The overlying epidermis was spongiotic and the stratum corneum demonstrated

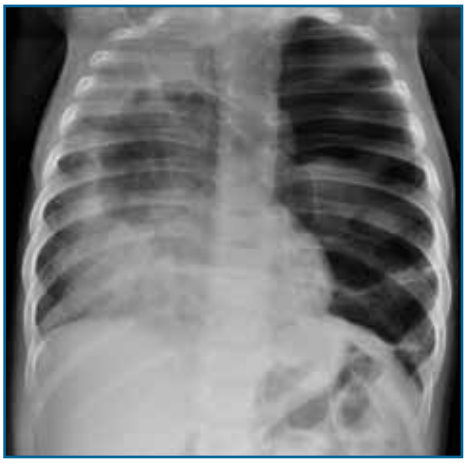

Figure 1: The initial chest X-Ray which demonstrates a pneumothorax on the left, with a diffuse opacity on the right side of the lung field

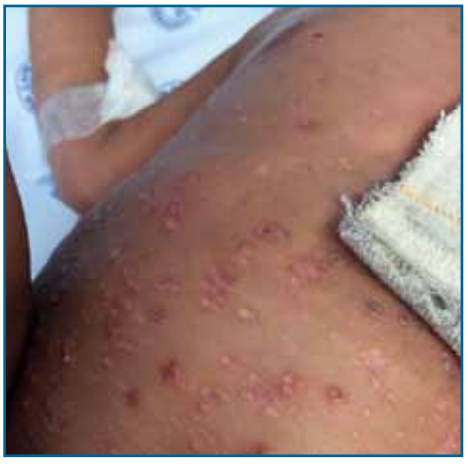

Figure 2: The diffuse rash that was initially treated as varicella zoster

hyperkeratosis, but ulceration was not seen.

A bone marrow trephine biopsy was very cellular and demonstrated morphological and immunohistochemical features suggestive of involvement by Langerhans cells/ Langerhans cell histiocytosis. The patient was commenced on chemotherapy which included prednisone and weekly vinblastine to which he showed a good initial response with regression of some of the skin lesions (Figure7). His clinical course, however, was complicated by recurrent pneumothorax on the left which required repeated intercostal drainage.

Full blood count (FBC)	White cell count: 19.56 x 109/L (n=6.00–18.00 x 10^9/L) Haemoglobin: 9.1 g/dL (n=10.1–12.9 g/dL) Mean corpuscular volume (MCV): 61.9 fL (n=70.0–86.0 fL) Platelet count: 521 x 109/L (n=140–35010^9/L) **Differential count** Neutrophils: 63.90 %; 12.50 x 109/L (n=2.00–5.50 x 10^9/L) Lymphocytes: 18.00 L%; 3.52 x 109/L (n=3.60–12.00 x 10^9/L) Monocytes: 17.90 %; 3.50 x 109/L (n=0.00–0.90 x 10^9/L) Eosinophils: 0.00 L%; 0.00 x 109/L (n=0.00–0.50 x 10^9/L) Basophils: 0.20 %; 0.04 x 109/L (n=0.00–0.20 x 10^9/L)
C-reactive protein (CRP)	5 mg/L (n=<10 mg/L)
TB gene Xpert	Negative
TB culture	Negative
Liver test (LFT)	Total protein=68 g/L (n=55–70 g/l) Albumin=35 g/L (n=28–48 g/L) Total bilirubin=5 µmol/L (n=5–21 µmol/L) Alanine transaminase=9 U/L (n=4–35 U/L) Alkaline phosphatase=179 U/L (n=82–383 U/L) Gamma-glutamyl transferase=15 U/L (n=1–39U/L)
Lactate dehydrogenase (LDH)	445 U/L (n=180–430 U/L)
Erythrocyte sedimentation rate (ESR)	7 mm/hr (n=0–10 mm/hr)
B-HCG (beta-human chorionic gonadotropin)	0 IU/L
Alfa-feto-protein	8.2 µg/L (n=0.6–28.3 µg/L)
Uric acid	0.33 mmol/L (n=0.09–0.39 mmol/L)
Blood culture	Negative
HIV Elisa	Negative

*Table 1: Relevant blood investigations (*n=normal value)*

Langerhans cell histiocytosis (LCH)

The presence of an anterior mediastinal mass with cystic lung disease, as in this child, is a rare presentation, however highly suggestive of Langerhans cell histiocytosis (LCH). LCH is a rare disease that results in monoclonal proliferation of the dendritic cell-related histiocytes.[1] The term eosinophilic granuloma has been used previously to describe local disease, whereas histiocytosis X, Letterer–Siwe disease, and Hand–Schüller–Christian disease have been used to describe the systemic form.[2]

LCH is part of a spectrum of histiocytic disorders that is classified into three classes.[3] Class 1, the dendritic cell disorders, includes LCH, secondary dendritic cell processes, juvenile xanthogranuloma, and solitary histiocytomas with a dendritic phenotype. Class 2 includes macrophage -related disorders and class 3 the malignant histiocytic disorders.[4]

Epidemiology

LCH is most common in children between one and four years of age with a peak incidence of about 0.1–1 per 100 000 children.[1] Males and females are equally affected with a higher frequency among Caucasians.[2,5] There are no genetic factors that affect the development of LCH and it is usually a sporadic illness.[2,5] A strong association between cigarette smoking and the development of pulmonary LCH has been described in adults; however, this association is not clear in children, neither is the effect of second-hand smoke exposure.[5,6]

Pathogenesis

In LCH, the histiocytes are morphologically and phenotypically similar to those of the Langerhans cells found in the skin as it expresses the same CD1a and CD207.[7-9] There is still a debate as to whether the clonal proliferation is due to a malignant transformation or is a result of an immunologic stimulus.[8,9]

Clinical features

LCH can be classified as single-system LCH (single organ involved) or multisystem LCH (two or more organs involved). In a retrospective analysis involving 1741 patients the following organs were involved: bone (77%), skin (39%), lymph nodes (19%), liver

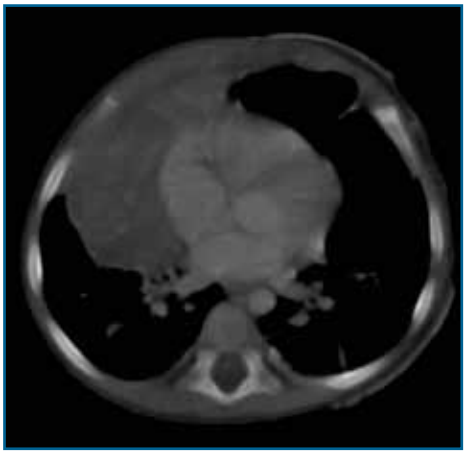

Figure 3: CT scan of the chest showing an anterior mediastinal mass

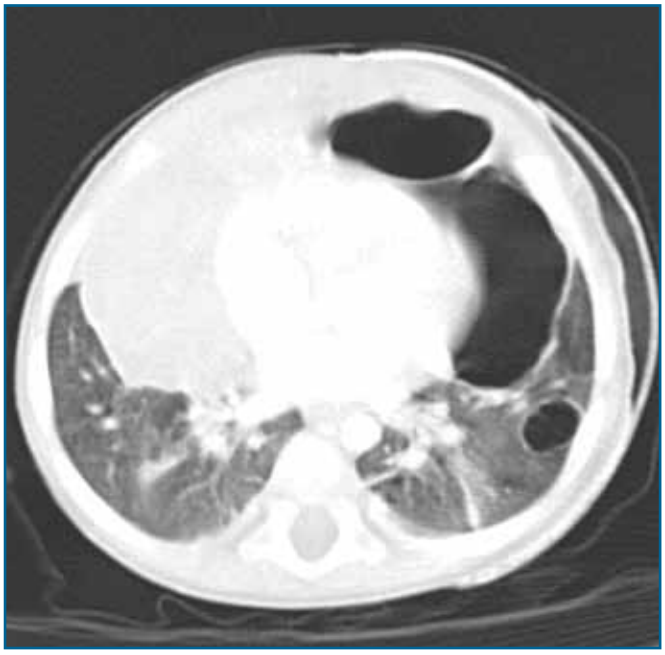

Figure 4: CT scan of the chest showing multiple cystic lesions of different sizes in the lung and a large anterior mediastinal mass

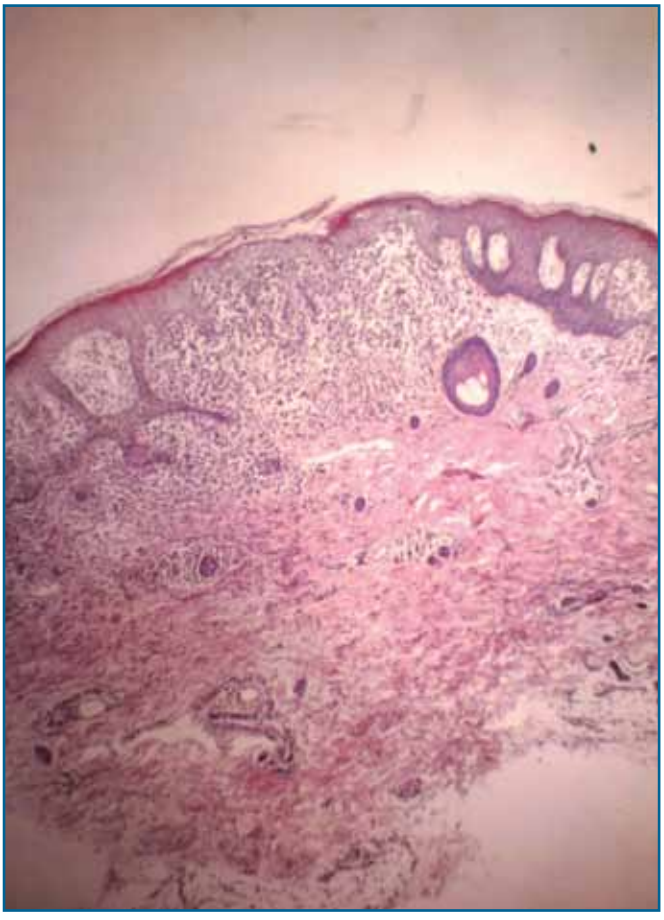

Figure 5: Low power of the punch biopsy showing a cellular infiltrate within the superficial dermis (Haematoxylin and eosin stain)

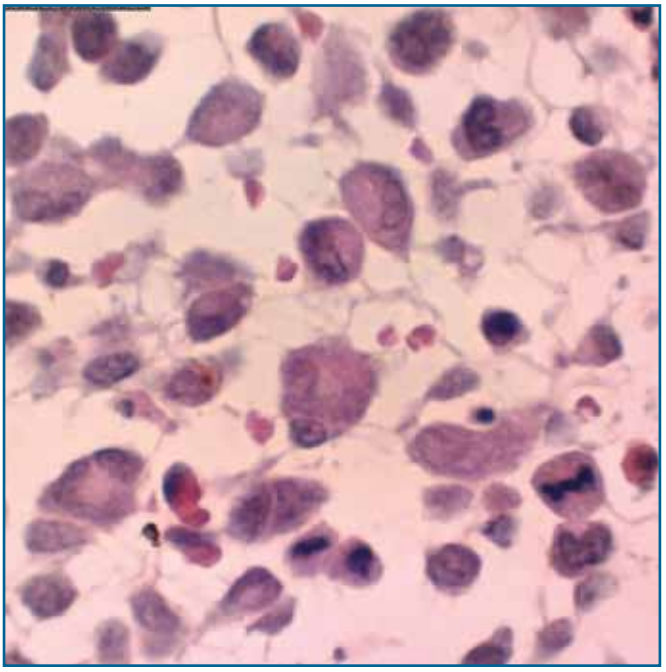

Figure 6: High power showing Langerhans cells associated with eosinophils (Haematoxylin and eosin stain)

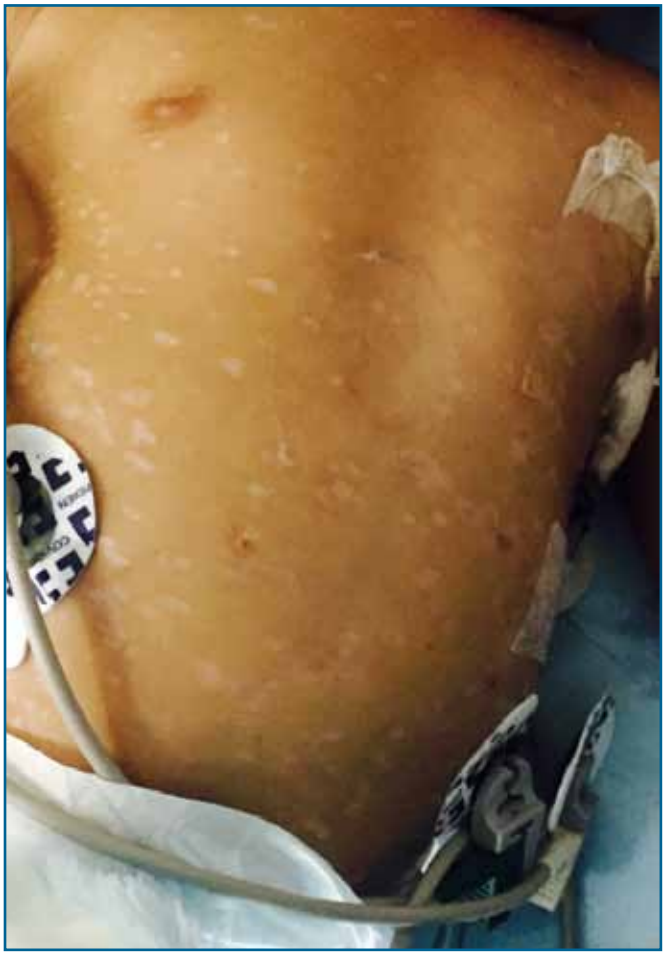

Figure 7: Skin lesions showing a substantial improvement on treatment (compared to Figure 2)

(16%), spleen (13%), oral mucosa (13%), lung (10%), and central nervous system (6%).[10]

Skin

In children under two years of age, cutaneous involvement is the most common presentation.[11] Cutaneous lesions can be extensive and can resemble seborrheic dermatitis and involve the face, trunk, and perineum. It may also present as papules, pustules, vesicles, petechiae, or purpura.[12] This was noted in our patient where this vesicular rash was initially thought to be varicella zoster and later a possible S aureus skin infection.

Lung involvement

Symptoms range from asymptomatic (with infiltrations noted on chest x-ray) to severe symptomatic respiratory disease.[6] These may be non-specific and include dyspnoea, cough, chest pain, wheezing, fatigue, wheezing, or tachypnoea. Chest pain may be associated with a pneumothorax.[1]

Mediastinal involvement

LCH with thymic and or mediastinal involvement is rarely reported. In a study with a large cohort of paediatric patients the frequency of mediastinal involvement was about 2.6% and the median age was 0.7 years.[13]

Diagnosis

The chest x-ray is usually abnormal and demonstrates a diffuse micro-nodular infiltrate or cytic changes (which are not common in children).[1,5] CT scan cysts may have a thick or thin wall and vary in size between a few millimetres up to 20cm. Involvement of the lower lung zones and costophrenic angles is noted in the paediatric population.[5] This was also noted in our child with the cysts involving the lingular of the left lung as well as the right lung.

Morphologic confirmation of pulmonary involvement can be obtained by bronchoalveolar lavage (BAL) or lung biopsy. The presence of Langerhans cells in BAL is identified by staining with antibodies against CD1a, and a proportion of CD1a-stained cells of more than 5% makes the diagnosis of pulmonary LCH likely.[2]

A skin biopsy can provide a quick and accessible way to make a diagnosis, and lung biopsy remains the gold standard to diagnose pulmonary LCH.[4] The CT scan is used to direct the area of the biopsy. This may be associated with sampling errors since the lesions of pulmonary Langerhans histiocytosis are focal.[2,5] A definitive diagnosis requires a positive staining with S-100 and CD1a antibodies and the presence of Birbeck granules on electron microscopy.[4] Electron microscopy however is not routinely used as immunohistochemistry is cheaper and more efficient.[3]

Treatment

Treatment protocols are based on whether there is single- or multi-organ involvement, as well as on the involvement of a high-risk (liver, spleen, hematopoietic organs) or low-risk organ (skin, bone, lymph node, pituitary gland, lung).[1,4]

No therapy is required in patients with limited cutaneous disease, however topical steroids are often used. Treatment for multi-organ disease is controversial, prednisone is sometimes used as a first line treatment, while others centres prefer the use of a single chemotherapy agent. Currently the protocol for the initial management of multi-system LCH include a six- week course of vinblastine and prednisone.[14] Newer therapies being investigated include monoclonal antibodies (targets CD1a or CD207), specific cytokine inhibitors, and 2-chlorodeoxyadenosine.[4]

Prognosis

Prognosis depends on the presence of high-risk organ involvement and a poor response to initial treatment. If both are present there is a 75% mortality rate, whereas those who respond to initial chemotherapy have a good survival rate.[1] Children who present at a younger age with multisystemic disease have a high mortality. Young age alone is not a risk factor for high mortality.[1]

Summary

The above patient provides a rare example of multi-organ (lungs, skin, and bone marrow) LCH presenting with an anterior mediastinal mass, cystic lung disease and typical skin features. In this case we illustrate the pulmonary features and complications caused by LCH in a young child. It also demonstrates the skin abnormality as one of the presenting signs, which may be misdiagnosed as eczema or cutaneous infections.

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Do conferences contribute to continuing medical education — or is it time for a change in approach?

J A Aluoch

Two apparently conflicting pieces of evidence exist about physicians' Continuing Medical Education (CME). Physicians report spending, on average (and among other activities), many hours per year in CME activities, ostensibly geared toward improving their performance and/or optimising the outcomes of their patients. In addition, producing and accrediting formal, planned CME events and activities are large enterprises intended to bring physicians up-to-date with rapidly expanding medical information. Patterned after undergraduate medical education consisting of lectures, audio visual presentations, and printed materials, CME activities appear underpinned by a belief that gains in knowledge lead physicians to improve how they practice and thus improve patient outcomes. Despite this belief and the level of participation in and resources dedicated to CME, many studies have demonstrated a lack of effect on physicians' performance of current practice guidelines or sizable gaps between potential and real performance. In addition, a relatively weak effect of formal, planned CME on physician performance has been demonstrated in some studies.

Despite seemingly endless rounds of conferences, symposia, round-table discussions, and panel debates over the years, CME now is not greatly different from what it was 40 years ago. There is simply a greater quantity of the same familiar things.

In light of the foregoing, one may be justified to ask: Why CME? Three generalisations keep recurring in the literature. We say, first, that it is the personal responsibility of professionals to engage in never-ending refinement of their professional competence; second, that the body of biomedical knowledge is changing so rapidly that each of us must struggle constantly simply to keep up with an increasingly narrow field since it is hopeless to try to keep abreast of general medical knowledge; and third, that many deficiencies in health care not only exist but could be corrected by the appropriate continuing education of practitioners—particularly those practitioners who do not take part in regular programmes of continuing education.

The diagnosis of deficiencies in the care of patients is surely an indispensable strategy, but far more difficult is the successful translation of even distasteful findings into sound educational practices that have some hope of alleviating the shortcomings which are identified. As professionals, we Doctors seem more willing to consider or even to adopt new information or new technology than to change in any fundamental fashion the way we use it ourselves. We are convinced, or so the literature of CME would make us seem, that it is our failure to apply new knowledge that represents the weakest link in the chain of assuring that the highest quality of medical care is delivered by the greatest number of physicians to the largest number of patients.

While this view may be correct, I am not familiar with any solid data to support it. In fact, the correction of the major health problems in Africa, as in other parts of the world, does not appear to require any substantial body of new knowledge. Rather, it requires that physicians use the knowledge they already have in a different way or more fully exhibit the professional attitudes that have characterised the physician's role as long as there have been physicians. As a more eloquent speaker recently put it, 'If I were asked to compose an epitaph on medical profession throughout the 20th Century, it would read: 'Brilliant in its discoveries, superb in its technological breakthroughs, but woefully inept in its application to those most in need….''

Since I was a medical student 50 years ago, I have heard and I have read in medical literature covering a far longer period that physicians can be of the greatest service to society if they work at preventing disease rather than treating it. But which gets more academic attention and reward: the replacement of damaged arteries and heart valves or the prevention of smoking and obesity? We have been told again and again that most of those who consult us are the anxious well rather than the curable sick. But which gets more attention in our educational programmes—the pharmacologic action of drugs and their side effects or the skill of listening and providing reassurance?

I am afraid that most of us have been seduced by the notion that we have a primary professional responsibility to keep abreast of current information—even if the information may have little use to many patients, and even if it means diverting attention from other elements of professional competence that may be of far greater importance to those we serve. Having been convinced that 'Keeping up' is the goal, we are easily led to the conclusion that the need in CME is for more instruction. Regrettably a recently completed survey by the World Health Organization on CME in member nations has shown that the lecture is still the most widely used instructional method by a large margin.

If, indeed, change in behavior is the goal of continuing education, whether it is offered to practitioners or to medical educators, then perhaps most of what we now do must be dismissed in

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much the same way as Oliver Wendell Holmes, the autocrat of the breakfast table and one-time dean of the Harvard Medical School, once dismissed another component of Medicine when he said: 'I firmly believe that if the whole materia medica as now used could be sunk to the bottom of the sea, it would be all the better for mankind—and all the worse for the fishes'.

It is time for change in our approach to Continuing Medical Education

The ultimate effect of formal CME interventions on the practice of physicians and the health of their patients—as in the case of any intervention—must be understood in the context of the methods by which the CME is delivered, including but not limited to the nature of the enabling resources available, the environment in which the translated competence is played out, and in the complex intrapersonal, interpersonal, and professional educational variables that affect the physician-learner's immediate goal of a CME activity. The exclusively didactic CME modality has little or no role to play. Knowledge is clearly necessary, but it is not in and of itself sufficient to bring about change in physician behavior and patient outcomes. Didactic interventions should receive less credit than do more effective methods—or perhaps they should receive no credit at all. In contrast, variables over which the CME provider has control and appear to have a positive effect are the degree of active learning opportunities, learning delivered in a longitudinal or sequenced manner, and the provision of enabling methods to facilitate implementation in the practice setting.

While numerous questions remain regarding formal CME, including group size, the role of the learning and practice environment, the clinical dimensions of care, the assessment of learner needs, and barriers to change, one question still looms large: In the face of longstanding knowledge about adult, self-directed learning and the general disinclination to believe that didactic CME works—now coupled with findings that indicate it does not—why would the medical profession persist in delivering such a product and accrediting its consumption? The reasons for the persistence of didactic CME include—but are definitely not limited to—the ease of designing and providing such activities, the substantial pharmaceutical sponsorship that promotes the transfer of information about new medications, and the dependence on traditional undergraduate models of education that are easy-to-mount and revenue generating.

Changing this delivery system carries serious implications for several groups of stakeholders that want to design and deliver effective CME. First, medical licensing boards and others with a genuine interest in assuring the public of physician competence must rethink the value of the CME credit system. Second, medical schools, specialty associations and societies, and other providers of CME must reconsider the value of the credit they provide, as well as the type and duration of learning activities they produce.

Further, organisations intending to ensure the quality of CME must evaluate the services that they provide to a large, complex, and expensive CME enterprise that values the production of single-session, teacher-centered activities over learner achievement. Finally, physicians must reflect on what they perceive as the CME experience itself and weigh the costs and lost learning opportunities of attendance at ineffective didactic sessions against participating in interactive, challenging, and sequenced activities that have enhanced potential for positively affecting their performance and the health of the patients they serve—the most important outcomes of all.

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Qualitative findings on the implementation of pulmonary rehabilitation in Kampala

J Pooler, R Jones, B Kirenga, W Katagira

Post-tuberculosis (post-TB) lung damage is irreversible and varies from mild to devastating in Uganda; it represents 20% of adult respiratory outpatient attenders. There is no useful treatment, but sufferers have poor health status and often are stigmatised.

The aim of this study was to adapt a UK model of pulmo nary rehabilitation (PR) for implementation in Kampala, Uganda, for patients with post-TB lung disease.

Working with Ugandan respiratory specialists, nurses and physiotherapists, we developed a PR team to design an exercise regime meeting international standards and an education programme which consisted of normal lung development, tuberculosis and post-TB damage, as well as conventional messages about breathlessness exercise, nutrition, smoking and drug treatments.

Between March 2015 — February 2016, we recruited a total of 45 patients to four x 6 week PR programmes. Each programme comprised 10–13 participants with only 10% drop-out. Interviews were conducted with 45 participants at baseline and six weeks post completion, and five stakeholder interviews. The purpose was to investigate the feasibility and acceptability of PR; impact of respiratory disease and PR; whether PR could be improved and the extent to which the exercises or gains were maintained.

Patients were debilitated by their condition before PR with fear of exercise. They reported whilst exercises were hard to complete at first, they noticed improvement in their ability to walk further and undertake work and domestic activity. As self-confidence improved, they felt less stigmatised and less depressed. Improved social and intimate relationships were reported and after completion, many continued exercises at home. Recommendations for future programmes included patient information to take home about the exercises, and to show their families; and the support of a community health worker to help maintenance of exercises at home.

PR is feasible and acceptable in post-TB patients in Uganda, but the programme must be culturally appropriate and education tailored to the patients' conditions. International collaboration worked very well, and as internet and skype was unreliable face-to-face meetings are essential. This study will inform the design of an implementation study planned for Zambia, Kenya, and Tanzania.

Use of Global initiative for asthma guidelines in asthma management among paediatric residents in tertiary hospitals in Nigeria

C Odimegwu, A Ayuk, A Ubesie, K Iloh

Although the Global initiative for asthma (GINA) guidelines provides a comprehensive integrated approach to asthma management that can be adapted for local use, non-adherence to such guidelines is one of the major reasons for poor asthma control.

A cross-sectional descriptive study of consenting pediatric residents in Nigeria using structured questionnaire. Questionnaire was adapted from GINA guidelines recommendations. Data analyses were with Statistical Package for Social Sciences (SPSS) version 19 (Chicago IL). Chi square was used to assess for any significant associations between categorical variables. A $p < 0.05$ was regarded to be statistically significant. All reported p-values were two sided.

Sixty-six paediatric residents aged 27–40 years were enrolled into the study (37 females and 29 males). An approximate one-third had spent more than three years in residency training. Fifty-eight residents (87.9%) were aware of the GINA guidelines while 46 (69.7%) were familiar with its contents. Only 39 (59.1%) residents adhered to the GINA guidelines. Twenty of the 35 junior residents (57.1%) compared to 26 of 31 (83.9%) senior residents were familiar with the GINA guidelines (p=0.031) while 15 of 35 junior residents (42.9%) compared to 24 of 31 senior residents (77.4%) consistently follow the GINA guidelines (p=0.006). Adherence to GINA guidelines was not influenced significantly by years of graduation or training (p>0.05).

The application of the GINA guidelines was poor among the interviewed paediatric residents. There is need for further exposure to asthma education and to incorporate the management guidelines into the paediatric residency training curriculum.

Asthma prevalence and risk factors in thirteen-fourteen year old school children in Lusaka, Zambia

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Zambia did not take part in the International Study of Asthma and Allergies in Childhood (ISAAC study) and, to date, country-specific data describing childhood asthma, allergic rhinoconjunctivitis and eczema do not exist. As such, the prevalence and risk factors of childhood asthma in Zambia are currently an unchartered territory. The present study sought to begin filling this knowledge gap.

School children aged 13–14 years were recruited as per ISAAC methodology. Inclusion criteria were all children in the Lusaka Urban District within the target age group. Children were asked to respond to (1) a standardised written questionnaire focused on wheezing, rhinitis, and eczema; and (2) a video questionnaire on wheezing.

A total of 1885 school children from 25 schools were included in the final study analysis. Nearly two-thirds (63.7%) of the pupils were aged 14 years and just over one-third (36.3%) were aged 13 years. There were slightly more girls than boys (56.2% vs 43.8%). The prevalence of current wheeze was 6.0%, and 8.2% of the children reported having been diagnosed with asthma by a health professional. Overall, 155 (4.3%) were found to suffer from severe asthma. Exercise-induced wheeze was present in 503 study participants (26.7%), and 35 (1.7%) children reported sleep disturbance due to wheezing. Night-time cough was reported by 661 children (35.1%).

The video questionnaire revealed prevalence rates of asthma symptoms in the last year to be as follows: wheeze 9.4%, exercise-induced wheeze 19.2%, sleep disturbance due to wheeze 5.3%, nocturnal cough 23.9%, and severe wheeze 8.4%.

A history of eczema or rhinoconjunctivitis was significantly associated with the risk of current asthma (adjusted odds ratios 3.48 [95% CI 2.17–5.58] and 2.38 [95% CI 1.57–3.63], respectively), self-reported asthma (adjusted odds ratios 1.92 [95% CI 1.18–3.12] and 2.10 [95% CI 1.45–3.12], respectively), and severe asthma (adjusted odds ratios 4.21 [95% CI 2.41–7.36], and 2.26 [95% CI 1.36–3.82], respectively).

Asthma prevalence in 13–14 year old school children in Lusaka, Zambia, is moderately high and comparable to rates found in other African major cities. Eczema and rhinoconjunctivitis are strongly associated with a diagnosis of asthma in Zambian children.

Prevalence of asthma and its symptoms among undergraduates in tertiary institutions in Ilorin, Nigeria

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Asthma is one of the world's most common long-term conditions,the prevalence and incidence for asthma throughout Africa has increased remarkably in recent years. There is paucity of data on the prevalence of asthma in Nigeria.The aim of this study was to determine the prevalence of asthma and its symptoms, self-reported asthma attacks, and use of asthma medication among tertiary institutions undergraduates in Ilorin, Nigeria.

The European Community Respiratory Health Survey (ECRHS) asthma-screening questionnaire was self -administered by participating students. Asthma was define as 'possible', current and physician diagnosed and compared with previous local studies to determine changing trend of asthma.

A total of 1485 students were enrolled into the study, with mean age (SD) of 20.9±3.1years and 793 (52.7%) were males. Nocturnal cough was reported by 21.4% (95% CI: 19.3–23.7%), shortness of breath by 13.5% (95% CI: 11.6–15.4%), and wheezing by 12.1% (95% CI: 10.5–13.9 %). Asthmatic attack in past 12-months was reported by 5.0% (95% CI: 3.9–6.1%), and current use of asthma medication by 7.5% (95% CI: 6.1–8.8%) of the respondents. The prevalence of possible asthma was 18.7% (95% CI: 16.7–20.7%), current asthma was 9.5% (95% CI: 7.9–11.1%) and physician-diagnosed asthma was 6.6% (95% CI: 5.5–8.0%). Possible, current and physician-diagnosed asthma and symptoms of asthma were higher in the females than the males. When compared to previous study in young adults ten years ago, the temporal trend shows an increase in the prevalence of asthma attack, use of asthma medication and physician diagnosed asthma.

The prevalence of asthma and its symptoms among tertiary institutions undergraduates in Ilorin, Nigeria is high and further buttressed the fact that asthma prevalence is on the rise.

The development and implementation of a Lung health programme for rural Uganda addressing biomass and tobacco smoke

R Jones, F van Gemert, B Kirenga

Chronic lung disease is common but under-reported in sub-Saharan Africa. Following a survey in rural Uganda which found 16% of the adult population had COPD, we developed a lung health awareness programme to detect and prevent chronic lung disease. This is a two year train-the-trainer programme conducted by health care workers (HCWs) led by the district health officer in Masindi district.

In this abstract we present the design and development of education programme and associated materials.

Working with HCWs who had conducted the Fresh Air Uganda survey, we implemented a train-the-trainer programme for HCWs who taught village health teams (VHTs) to teach their communities. We held a series of meetings to develop the project strategy and contents of the education materials. Preliminary education materials were shown to senior clinicians, administrators (including the Minister for Health and the District Health Officer in Masindi) through all grades of clinicians to VHTs and villagers. Incorporating all feedback in an updated programme, the first group then taught other HCWs and again adapted the materials. Final educational materials covered: 'What is lung health?', 'How the lung gets damaged?', 'Lung growth and development', and 'Preventing harm by reducing exposure to tobacco smoke and biomass smoke'.

We designed radio messages for broadcast on talkshows and radiospot adverts locally.

Evaluation methods included designing knowledge questionnaires for use before and after training for HCWs and the population.

Educational materials for use in training HCWs and VHTs using desk-aid flip-over charts, and posters have been designed and approved by the Ministry of Health.

The target was to train 10 HCWs in the first group and 12 completed this. These 12 then trained 47 HCWs, and VHT training is ongoing with over 100 so far completing. We have developed and administered knowledge questionnaires.

Using a ground upward approach we involved the local health care systems and HCWs to develop and deploy a train-the-trainer programme which is continuing. The educational materials were designed by the team are now approved for national use. The materials are available for wider use including in our international projects, in pulmonary rehabilitation and in midwifery training.

Asthma and pneumonia among children less than five years with acute respiratory symptoms in Mulago hospital, Uganda: evidence of under-diagnosis of asthma

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Pneumonia is considered the major cause of mortality among children with acute respiratory disease in low-income countries but may be over-diagnosed at the cost of under-diagnosing asthma. We report the magnitude of asthma and pneumonia among 'under-fives' with cough and difficulty breathing, based on stringent clinical criteria. We also describe the treatment for children with acute respiratory symptoms in Mulago hospital.

We enrolled 614 children aged 2–59 months with cough and difficulty breathing. Interviews, physical examination, blood and radiological investigations were done. We defined asthma according to Global Initiative for Asthma guidelines. Pneumonia was defined according to World Health Organization guidelines which were modified by including fever and, white cell count, C-reactive protein, blood culture and chest x-ray. Children with asthma or bronchiolitis were collectively referred to as 'asthma syndrome' due to challenges of differentiating the two conditions in young children. Three pediatricians reviewed each participant's case report post hoc and made a diagnosis according to the study criteria.

Of the 614 children, 41.2% (95% CI: 37.3–45.2) had asthma syndrome, 27.2 % (95% CI: 23.7–30.9) had bacterial pneumonia, 26.5% (95% CI: 23.1–30.2) had viral pneumonia, while 5.1% (95% CI: 3.5–7.1) had other diagnoses including tuberculosis. Only 9.5% of the children with asthma syndrome had been previously diagnosed as asthma. Of the 253 children with asthma syndrome, 95.3% (95% CI: 91.9–97.5) had a prescription for antibiotics, 87.7% (95% CI: 83.1–91.5) for bronchodilators and 43.1% (95% CI: 36.9–49.4) for steroids.

Although reports indicate that acute respiratory symptoms in children are predominantly due to pneumonia, asthma syndrome contributes a significant proportion. Antibiotics are used irrationally due to mis-diagnosis of asthma as pneumonia. There is need for better diagnostic tools for childhood asthma and pneumonia in Uganda.

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