CURRENT CONCEPTS IN THE DIAGNOSIS AND MANAGEMENT OF CHILDHOOD ASTHMA

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Zulfiqar Ahmed Bhutta (Department of Paediatrics, The Aga Khan University & Medical College, Karachi.)

INTRODUCTION

Asthma, one of the commonest childhood disorders, is frequently underdiagnosed and inappropriately treated. Its diagnostic criteria have undergone considerable revision and definition recently, there is evidence that both the prevalence and severity of asthma are increasing[1,2] in the west, It is possible that environmental factors may be responsible for this change[3] and several studies from the developing world have also implicated environmental changes in the apparent increase in asthma prevalence[4,5].
There is a dearth of published data on childhood asthma in Pakistan although it is an ubiquitous problem and may account for over 30% of all outpatient referrals[6]. Most such children are significantly incapacitated by the disorder, and a hospital based study found moderate to severe asthma in over half of the children studied[7]. It is also clear that despite diagnosis, most cases of childhood asthma are frequently under-treated.
The purpose of this paper is to review some of the recent advances in the diagnosis and management of childhood asthma.

PATHOPHYSIOLOGY

Although a variety of definitions are employed, a common physiological definition is of a “disease cnaractensed by hyper-responsiveness of the airway to various stimuli, resulting in airways obstruction that is reversible either spontaneously or as a result of treatment”[8].
The airways of an asthmatic child are unduly “twitchy”. Irrespective of the initial triggering factor, the airways may respond to a stimulus by bronchoconstniction due to spasm of bronchial smooth muscles and an inflammatory response leading to mucosal edema and increased secretions.
The end result is considerable airflow obstruction, mucus plugging, ventilation-perfusion mismatch and consequent hypoxia. In recent year, the recognition of the inflammatory response in asthma has led to significant alteration in the approach to therapy[9].

DIAGNOSIS

The diagnosis of childhood asthma is usually suggested by the history. A history of nocturnal cough, occasionally paroxysmal and associated with vomiting, is frequently elicited in children. Although intermittent wheezing is common, it is episodic and may not be actually present at the time of examination.
In younger children (under 5 years) unable to perform lung function tests, the diagnosis is usually clinical and confounded by the fact that wheezing may be seen in response to a variety of viral infections. The commonly employed term “wheezy bronchitis” has been used to denote such children who have recurrent episodes of wheezing and exacerbation of cough in association with viral infections. Indeed viruses have been isolated in upto 42% of all children with acute wheezing episodes10. Such wheezing in response to viruses occurs in 10 - 20% of all infants and young children[11]. The term “wheezy bronchitis” was coined to denote such infants and young children who

only exhibited wheezing in response to viral infections and outgrew their symptoms during their preschool years. However, the term should be avoided as it detracts from the early diagnosis of many asthmatic children and leads to under-treatment[12].

Recent evidence indicates though that there may be at least two patterns of childhood asthma in infancy[13]. There are a group of children with wheeze associated with viral infections, who come from non-atopic backgrounds and outgrow their symptoms. In contrast there are children with a clear atopic background, often associated with eczema and a strong family history, who develop symptoms which persist until late childhood. It may be impossible to distinguish between the two at times and there is considerable overlap in presentation[14]. The treatment, however, of bothgroups with wheezing in early childhood is fairly similar with only prognostic differences.

The diagnosis of asthma in older children, who can perform lung function tests, is simpler. Simple lung function tests such as peak flow rate (PFR) using an inexpensive portable peak flow meter (Mini-Wright's Peak Flow Meter-Approx: Cost Rs. 900.00) and forced expiratory volume in one second (FEW) can be easily performed in an office setting and, in conjunction with clinical symptomatology, provide a reasonable accurate confirmation of diagnosis. Standard height related PFR charts are available for quick comparision[15]. The follow. ing lung function criteria are used to support the diagnosis of asthma[13]:-

1. Excessive variability of PFR or FEW over short periods of time, defined as a fall of at least 20% from the established maximum for any given child.
2. Improvement of at least 20% in PFR or FEW after administration of an inhaled B2 stimulant.
3. Decrease of >20% in PFR or FEW after bronchial challenge (usually easily provided by an exercise test).

Although an allergic basis or IgE mediated reaction has been shown to occur in many cases of asthma[16], the role of allergy testing in the diagnosis and management of childhood asthma remains controversial. Total IgE estimation may provide useful additional information, but there is little to recommend routine skin testing or specific bronchial challenge with allergens.

MANAGEMENT OF ASTHMA

The common management goals in childhood asthma are reduction in symptoms and returning the child to normal activity, employing treatments with minimal side effects. Although a great deal of attention is focused on drug therapy of asthma, other aspects of therapy and counselling of the family are equally important. Adequate attention must also be paid to the emotional effects of asthma on the growing child.

a) Regular monitoring: It is crucial that a base-line record of severity of clinical symptoms and some form of lung function measurement (preferably home PFR measurements), be maintained. A diary-card with an accurate record of symptoms, PFR, need for therapy and general well-being is of incalculable help in planning and maintaining therapy. Such parental involvement in therapy also serves the purpose of educating the child and the family in self management.

b) Environmental manipulation: Although an allergic component is evident in a number of children with asthma, a specific trigger can only bc identified in a minority of cases. If so, such triggering factors should be avoided, Measures can be adopted to reduce exposure to seasonal pollen, sudden changes in environmental temperature and humidity. A large proportion of children with asthma show sensitisation to house dust mite and some simple measures can he adopted to decrease exposure to it. These include wet dusting, avoidance of thick carpets and cotton filled mattresses and pillows. Psychotherapeutic measures can be helpful when emotional factors are considered significant in

triggering asthma.
c) Phannacotberapy: Drug therapy remains the mainstay of asthma management and is an area in which considerable advances have been made over the years, particularly in better agents and delivery systems. With a clearer understanding of the role of inflammatory triggers in chronic asthma, it is evident that therapy must also control inflammation in addition to bronchodilatation[17].

TABLE I. Drugs used in the treatment of asthma.

	Parenteral Administration	Oral Administration	Aerosol Administration
A. Sympathomimetics Selective B2 agonists Salbutamol (Albuterol) (Ventolin)	IV Bolus: 10 mcg/kg over 10 min. Maintenance: 0.2 mcg/kg/min, can be raised by 0.1 mcg/kg q 15 min to a maximum of 4 mcg/kg/min. (if tolerated)	2 mg per 5 ml syrup, 2 mg or 4 mg tablets kg/dose, 3-4 times daily upto 4 times daily	0.5% solution: 0.01 to 0.03 mg/kg to a maximum of 1 ml diluted with saline to 3 ml In acute severe asthma may be used every 20 min in hospital, until a clinical response occurs.
Terbutaline	Subcutaneous 0.005 mg/kg q 30 min x 3 prn (max 0.5 mg in 4 hrs.)	300 mcg/ml syrup, 2.5 or 5 mg tablets 0.075 mg/kg/dose, 3-4 times daily	1% solution: 0.03 ml/kg/to a maximum of 1 ml diluted with saline to 3 ml, upto 4 times daily
Non-selective sympathomimetics Epinephrine (Adrenaline)	1: 1000 soln, 0.01 ml/kg SC q 20 min x 3 prn (max 0.4 ml)		
B. METHYLXANTHINES Theophylline Slow release preparations		20 mg/kg/ day q 6-12 hourly (some children may need more than 20 mg/kg/day)	
Aminophylline	IV 6mg/kg stat, followed by continuous infusion 0.8-1 mg/kg/hr	Dose conversion (0.85 x Theophylline)	
C. STEROIDS Prednisolone Hydrocortisone	IV 4 mg/kg stat, followed by continuous infusion at 1 mg/kg/hr	1-2 mg/kg/day q 12 hourly or 24 hourly	
Beclomethasone			400-800 mg/kg/day q 6-12 hourly
D. DISODIUM CROMOGLYCATE (Intal)			Spinhaler 20 mg q 6 hourly Inhaler 2 mg q 6 hourly
E. KETOTIFEN		2.5 - 5 ml BD	

Table 1 lists some of the common drugs and dosages employed in the treatment of asthma.
The principles of therapy are to provide tailor-made regimen according to the severity of asthma. The response to therapy can also assist in determining the severity of asthma. It is useful therefore, to consider therapy accordingly, and the following classification is clinically useful.
i) Mild asthma Discrete attacks occurring less than once a month, or as more frequent minor
ii) Moderate asthma. Discrete attacks occurring no more frequently than once a week, and in which no more than two to three weekly doses of bronchodilators are required.
iii) Severe asthma. Attacks occur more than once a week with occasional poor response to bronchodilators.

AMBULATORY MANAGEMENT OF ASTHMA

In general, mild asthma can be treated with B2 agonists given as required. Moderate asthma requires more frequent B2 agonist therapy with additional anti-infiarnmatory agents such as disodium cromoglycate for prophylaxis. In younger children, where inhaled therapy may be difficult, oral xanthines may be required. However, there are significant side effects to long term xanthines therapy including irritability, insomnia, poor school performance and abdominal discomfort. It is also difficult

to titrate the dose of xanthines because of interaction with drugs such as erythromycin and alteration of levels with a variety of viral infections. In addition, the therapeutic margin of xanthines is very narrow and many children may show unacceptable side effects despite therapeutic levels of the drug (10-20 mg/I).

TABLE II. Manifestations of severe asthma.

* Cyanosis
* Altered mental status
* Inability to speak
* Profuse diaphoresis
* Minimal relief with bronchodilator therapy
* Absence of wheezing ("silent chest")
* Respiratory rate >30/min
* Pulsus paradoxus >15 mmHg
* Use of accessory muscles of respiration
* Peak flow rate <20% predicted
* PaO_2 <60 mmHg (room air)
* $PaCO_2$ >40 mmHg
* Pneumothorax / Pneumomediastinum

More severe attacks (Table II) require in-haled corticosteroids and such children may also need additional sustained release xanthines preparations. A major change in recent years is the preferential early use of steroids in moderate to severe asthma.

In general, inhaled therapy is preferred at all ages, although a homenehuliser or spacer device may be needed for younger children. This reduces the unacceptable side effects of oral B2 agonists and may obviate the need for oral xanthines with their familiarity with inhaler or myriad side effects. Lack of nebuliser therapy is a major reason for reliance on oral drugs by many primary care physicians. However, oral therapy requires nearly 20 times the inhaled dose for a comparable effect and is thus associated with considerable side effects, Inhaled corticosteroids, when used in doses under 600 mcg/day, do not cause significant adrenal suppression and are usually employed in the minimal dose necessary to control symptoms.

The decision to initiate prophylaxis is based on a variety of factors. These include frequent attacks (>6 per year) necessitating school absenteeism, infrequent but severe attacks and development of chest deformities. Inhaled or nebulised disodium cromoglycate has been shown to be an extremely effective prophylactic in childhood asthma A commonly used alternative prophylactic, ketotifen, has been shown to be efficacious in the treatment of atopic eczema and allergic rhinitis but its benefit in childhood asthma is controversial[18].

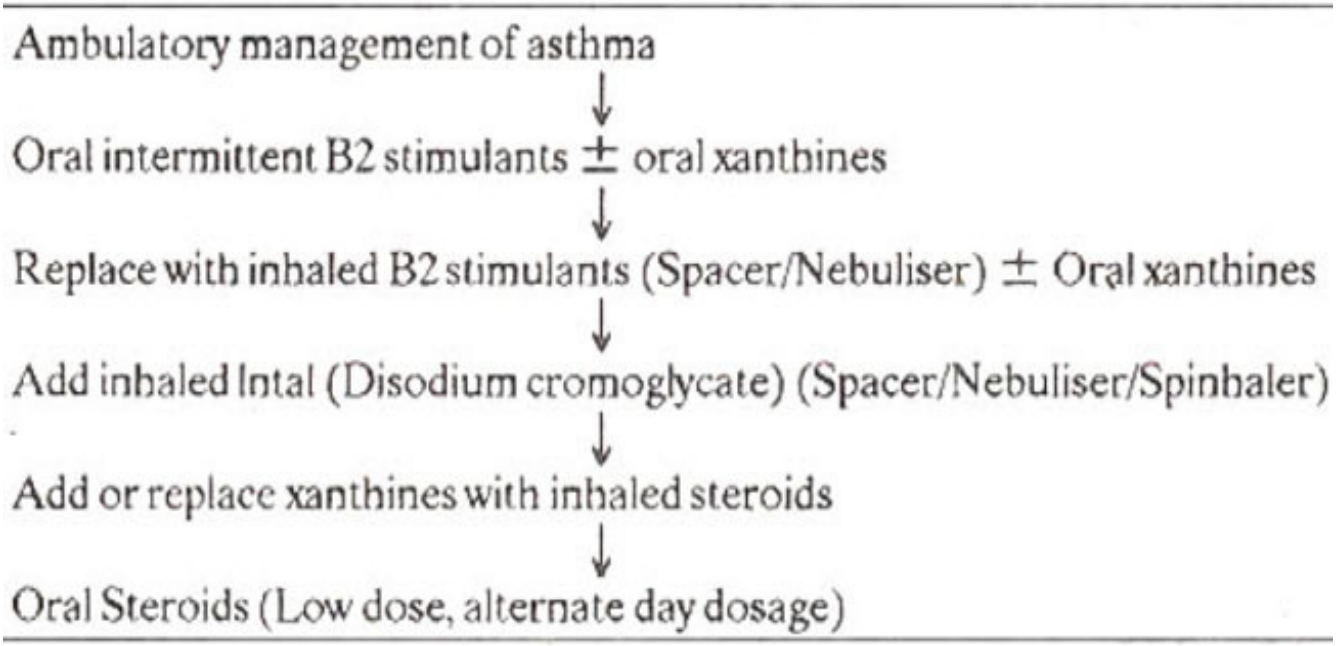

Figure 1. Ambulatory management of asthma.

Figure 1 suggests a scheme for the ambulatory management of moderate to severe asthma. Appropriately used inhalation therapy forms the mainstay of asthma therapy and adequate time must be spent by the treating physician in explaining and observing inhalation technique. In younger children a spacer device may be used to administer inhaled medication, obviating the need for synchronized respiration. A simple spacer device can be fashioned using an ordinary polystyrene cup with a hole cut in the bottom to accommodate the inhaler and upto ten puffs can he given at one time. Adequate explanation of the need for therapy to parents and assurance of the lack of "addictive properties of inhaler therapy is essential to ensure compliance. In infants and young children it may be necessary to use a portable nebuliser (Portable Nebuliser Electricity operated. Various types. Average cost Rs. 5000-5500) and this device should now be considered an essential equipment for a paediatric clinic.

Of the various causes of failure of therapy, the majority are related to poor compliance, inhalation technique and/or inappropriate dosage. In some intractable cases, chronic sinusitis or unrecognized gastroesophageal reflux may be responsible for recurrent attacks and poor response. In addition, the importance of psychosocial factors in the difficult adolescent asthmatic should not be underestimated and may be the main underlying cause in a problematic patient.

MANAGEMENT OF THE ACUTE ATTACK OF ASTHMA

The therapy of an acute exacerbation of asthma or a severe attack requires a keen awareness of the potential life threatening nature of the disorder. Improperly recognized and treated, the state can lead to a vicious cycle of progressive hypoxia, dehydration, acidosis and respiratory failure. The most important measures in rapid evaluation are clinical observation of oxygenation, neurological status and signs of exhaustion. As immediate drug therapy is instituted, close attention must also be paid to

adequate hydration and oxygenation. With the development and availability of means of nebulised therapy, these have assumed first line status in the management of acute asthma. Even though nebuliser solutions may not be available, injectable solutions can be nebulised using an ordinary source of oxygen and simple nebuliser cup (Hudson nebuliser cup-Approx price Rs. 70.00 Catalogue number 1710/1713). However, should they not be available, subcutaneous adrenaline may still have a role. A suggested scheme for the management of an acute attack is shown in Figure 2.

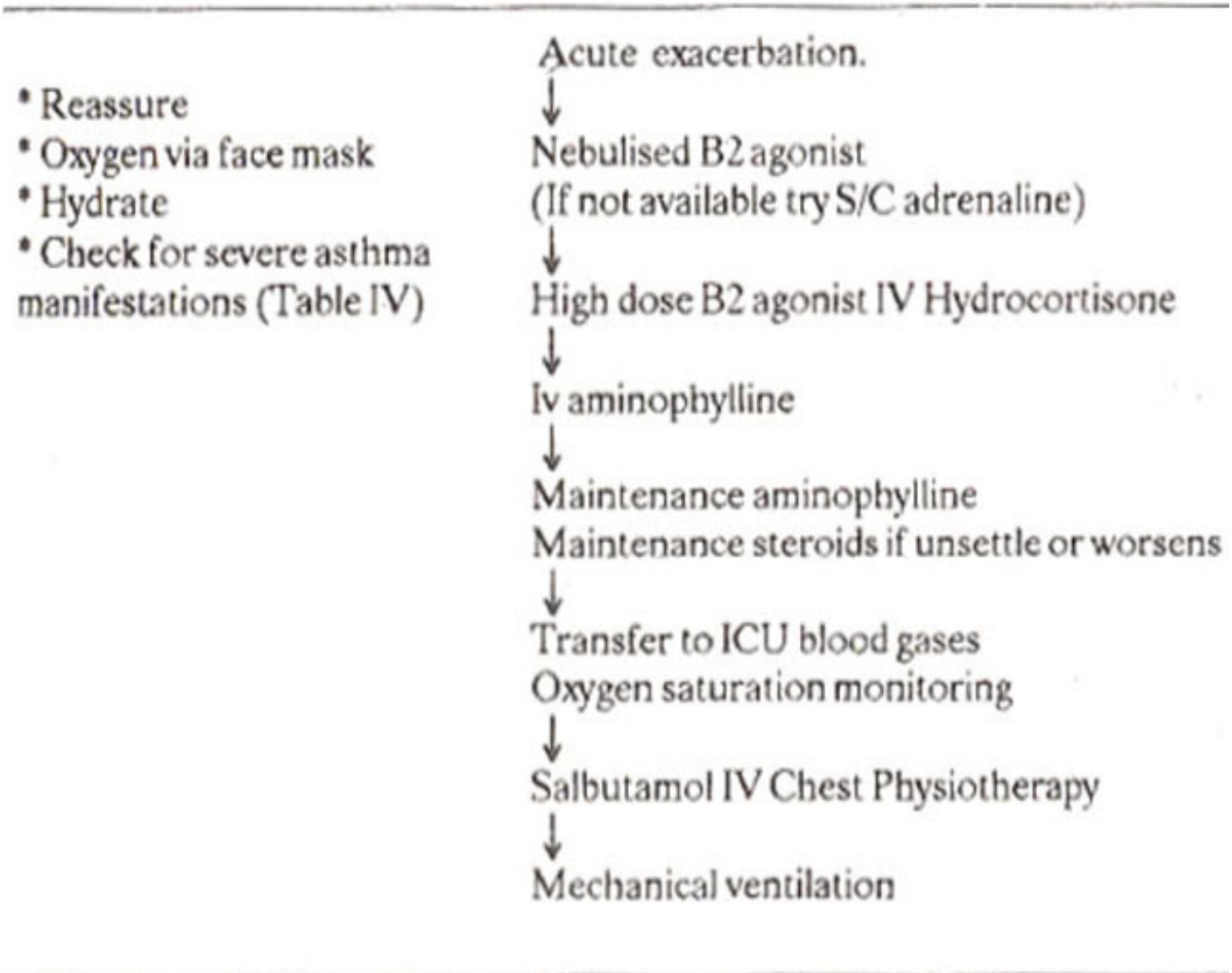

Figure 2. Acute exacerbation.

As evident, the tendency now is to use steroids early in the course of an acute attack. It is also important to place the child on adequate maintenance therapy after stabilisa¬tion in the emergency room. If the child fails to satisfactorily respond to bronchodilators, further therapy should be continued in an intensive care unit with close monitoring of vital parameters and blood gases. Rarely, paralysis and ventilation may be required. If so, a volume ventilator with servo control may be preferable to other forms of ventilation.

An acute severe attack must always suggest failure of prophylaxis, and close followup and adjustment of therapy after discharge is mandatory.

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