Dental treatment in children with asthma – a review

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IN BRIEF

- Provides an understanding of the systemic effects of long-term use of asthma medications.
- Highlights the effects of asthma and its associated medications on the oral cavity.
- Discusses considerations for the general dental practitioner when treating asthmatic patients.

GENERAL

This article aims to explain the effects of long-term use of asthma medications. It will discuss the effects of asthma on the oral cavity, as well considerations for the dentist when treating asthmatic patients. It will also explain how to manage asthma in the dental setting and provide advice on maintaining oral health for asthmatic patients.

INTRODUCTION

Asthma is one of the most common chronic diseases of childhood affecting 1.1 million children in the UK (1 in 11).[1] Worldwide prevalence is estimated at 300 million, a prevalence that continues to increse.[2] Asthma is defined as a chronic inflammatory condition characterised by variable and reversible episodes of airflow obstruction and bronchospasm. Symptoms include wheezing, shortness of breath, chest tightness and coughing, often in response to an identifiable trigger. Asthma is a cause of considerable morbidity worldwide. It is estimated, however, that as much as 75% of hospital admissions for asthma are avoidable and as many as 90% of deaths are preventable.[1]

ASTHMA CLASSIFICATION

Historically, asthma was classified according to severity of symptoms, but more recently the Global Initiative for Asthma (GINA) has classified asthma both according to disease severity and adequacy of symptom control.[3] This represented a shift in asthma classification and indicates the recognition that asthma severity is both a manifestation of underlying disease severity and responsiveness to medication. GINA classifies asthma as controlled, partly controlled or uncontrolled. In essence, a patient with controlled asthma should experience minimal symptoms, no exercise limitation, minimal requirement for rescue medication, near-normal lung function and the condition would be exacerbated infrequently.[4] Significantly, these are questions that are easily elucidated by the general dental practitioner (GDP) and should be noted at each dental visit.

ASTHMA AND MEDICATIONS

The majority of asthmatic patients will require a form of long-term 'preventive' medication usually in the form of inhaled corticosteroids (ICS) in order to prevent exacerbation of their symptoms. Sustained bronchodilatation may be achieved by the use of inhaled long-acting beta-2 agonists. Increased symptoms may be treated by shorter acting medications including inhaled beta-2 agonists or anti-cholinergic drugs. Additionally, depending on asthma severity other agents such as inhaled mast cell stabilisers, oral anti-leukotriene agents, systemic corticosteroids, and newer monoclonal antibodies may be utilised to attain control of symptoms.[5]

Asthmatic patients may be at an increased risk of significant adverse reactions to commonly used medications. It has been reported that 7% of patients with asthma and 14% of patients with severe asthma have aspirin-exacerbated respiratory disease. In severe cases the reactions can lead to intense bronchospasm and fatal anaphylaxis.[6]

Cross-sensitivity has been noted between aspirin and other non-steroidal anti-inflammatory drugs (NSAIDs) and, therefore, therapy with NSAIDs should be avoided in asthmatic patients with a history of aspirin or other NSAID sensitivity. Equally, it should be administered cautiously in all patients with pre-existing asthma, whether or not they have a history of sensitivity.[7] Aspirin desensitisation has been shown to be effective and can lead to patients overcoming their allergy to aspirin and NSAID drugs, with a subsequent reduction in rescue courses of oral corticosteroids to control their symptoms.[8]

Opiate drugs are also contra-indicated in severe asthmatic patients as they can lead to respiratory depression and histamine release, therefore precipitating a severe attack.[9] Due to the above indications, paracetamol is the drug of choice for pain relief in asthmatic patients.

Dental antibiotic prescribing in asthmatics has not been found to have many contra-indications and most antibiotics do not interfere with asthma treatment. Patients taking theophylline-containing asthmatic medications should avoid erythromycin and other macrolide antibiotics as they interfere with theophylline metabolism and can lead to toxic levels of methylxanthines in the blood.[10]

Inhaled corticosteroids are commonly prescribed as a preventive medication and side effects are principally local, compared with the oral or parenteral route. However, systemic effects are seen with high-dose therapy. These adverse effects may include the suppression of the hypothalamo-pituitary-adrenal axis, thinning of the skin, stunted growth in children, ophthalmic effects and osteoporosis.[11] This is treated with vitamin D and calcium supplementation, bisphosphonate therapy or the administration

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Refereed Paper
Accepted 22 February 2016
DOI: 10.1038/sj.bdj.2016.220
©British Dental Journal 2016; 220: 299-302

of monoclonal antibodies. Patients on bisphosphonate therapy may be at an increased risk of bisphosphonate related osteonecrosis of the jaw (BRONJ), especially those on intra-venous bisphosphonates.[12] These patients require careful treatment planning and liaison with the patient's physician, particularly if dental extractions are required.[13] Extractions should be completed as atraumatically as possible and if it is a particularly difficult extraction, a referral to a specialist oral surgeon or maxillofacial team may be appropriate. There is currently no evidence to support antibiotic or topical antiseptic prophylaxis in the prevention of BRONJ.[14]

DENTAL CARIES

There has been considerable debate in the literature regarding the caries risk of asthmatic patients. Studies have demonstrated higher caries prevalence in both the primary and permanent dentitions of children with asthma.[15,16] Conversely, alternative research has failed to demonstrate an association between dental caries and asthma.[17] A recent systematic review has concluded that asthma doubles the risk of dental caries in both the primary and permanent dentition.[18] Numerous theories have been postulated to explain this increased caries risk including increased medication effects, increased prevalence of mouth-breathing, increased consumption of sugary drinks and the risk of parental overindulgence. Therefore, it is likely that the aetiology is multifactorial.[19]

Saliva has a beneficial effect on caries reduction through four main actions: physical cleansing, antibacterial effect, buffering capacity and super saturation with calcium phosphate. Prolonged use of a beta-2 agonist can significantly reduce salivary flow. Rybery *et al.* reported that overall salivary flow decreased by 26% and parotid flow reduced by 36% in asthmatic groups on medication, when compared with controls.[20] This group also determined that asthmatic patients have a reduced salivary output per minute of amylase, lysozyme, salivary peroxidase and secretory IgA in stimulated salivary flow.[21] Therefore, reduced quantity of saliva, combined with a reduced quality of secreted saliva may significantly increase caries risk for asthmatic patients. In addition, Kargul *et al.* described a decrease in salivary and plaque pH to below the critical value of 5.5, 30 minutes after the use of beta-2 agonist inhalers.[22] This has been confirmed in a study by Tootla *et al.*, who found that dry powder inhalers consistently produce a pH below 5.5 indicating their cariogenic potential.[23] Certain inhalers contain fermentable carbohydrates in the form of lactose. This aims to mask the bitter medication taste and improve patient tolerance, but may contribute to an increased caries risk. Asthmatic patients have also been found to show increased counts of the cariogenic bacteria *S. mutans*.[20,21]

In addition, asthmatics have a significantly increased risk of nasal polyposis which may predispose them to mouth breathing.[24] The prevalence of developmental defects of enamel is reported to be increased in the asthmatic patient with its consequent risk of caries. Guergolette *et al.* estimated an 11-fold increased risk of enamel defects compared with controls. This correlated with increased disease severity and earlier symptomology.[25]

Decreased salivary flow as a result of regular beta-2 agonist inhalation, combined with consistent mouth breathing, can create a feeling of thirst. To compensate for this oral drying, asthmatic patients may drink regularly. The continual masking of the bitter medication taste and thirst quenching with carbonated drinks, combined with reduced salivary protection and increased prevalence of enamel defects, may be a major contributor to caries amongst these patients. The long-term use of sugary medications, including antibiotics and analgesics, which may occur in the asthmatic population has been shown to be deleterious to dental health.[26]

DENTAL EROSION

It has been suggested that asthma medication and its side effects contribute to dental erosion. Saliva is the main neutraliser of daily dietary acids in the oral cavity. This increased risk of dental erosion may be attributed to the reduction in salivary flow as mentioned previously.[21] Tootla *et al.* measured the erosive potential of inhalers. Those with a higher titratable acidity are considered to have a higher erosive potential for dental tissues. Despite the fact that asthma inhalers, particularly lactose-based inhalers, produce a low oral pH, their titratable acidity has not been shown to have an erosive potential for dental tissues.[23]

An increased risk of dental erosion may be explained by the increased incidence of gastro-oesophageal reflux disease (GORD) amongst asthma sufferers. GORD occurs when gastric acid within stomach contents escapes into the oesophagus. The most common symptoms include heartburn and acid regurgitation, but consequences of reflux may be more extensive. An association between GORD and asthma has been accepted for over a century since Osler's comment that 'severe paroxysms of asthma may be induced by overloading the stomach.'[27] GORD is 75% more prevalent in asthmatic patients when compared with controls.[28] However, it is unclear whether there is a causal relationship between asthma and GORD, or alternatively, whether this represents a chance association between two common childhood conditions. There are a number of possible explanations for this increased prevalence. The 'reflux theory' suggests that the bronchial tree is damaged as a result of occult acid inhalation, whereas the 'reflex theory' postulates that bronchospasm is precipitated by acidic stimulation of vagal nerve endings in the oesophagus.[29] Additionally, acid reflux may be exacerbated by the increased respiratory effort of coughing, which increases the pressure gradient across the abdomen. Al-Digan *et al.* suggested that GORD may be caused by partially swallowed beta-2 agonist medication, resulting in lower oesophageal sphincter pressure and oesophageal contraction amplitude. This relaxation is associated with GORD.[30] A significant association between asthma and GORD has been reported in the literature, although the direction of causality remains unclear.[31]

To minimise the effects of medication and help prevent dental erosion, patients can be encouraged to rinse with water or fluoride mouthwashes following administration of medication in order to neutralise oral pH. The use of a spacer device to direct the acidic medication directly into the lungs may help. Patients are also advised to brush twice daily with a fluoride toothpaste.[28,32] As there is a strong association between asthma and GORD, all dentists must be proactive at making appropriate referrals if tooth erosion is noted.

PERIODONTAL DISEASE

Studies investigating the association between asthma and periodontal disease have shown equivocal results.[5] It has been suggested that the relationship between the two may be attributed to either the side effects of the asthma medication, pathological activation of the inflammatory and immune process or a combination of the two. The protective mechanisms within saliva balance the interactions between bacterial and immunological factors and help maintain periodontal health. A decrease in salivary flow and secretory IgA, caused by long term use of asthma medication, reduce its protective qualities and therefore place the patient at greater risk of developing periodontal disease. In addition to this, studies have reported that gingival tissues of asthmatic patients have markedly elevated IgE levels, which are responsible for periodontal destruction.[33]

Oral hygiene has been shown to be poor in the asthmatic population. McDerra *et al.* reported higher plaque deposits in 4–10-year-old asthmatic children, compared

with controls. This was not replicated in the 11–16 year group. It was suggested that parents of the younger age group did not consider dental health to be a high priority, compared with the asthma diagnosis. Increased levels of gingivitis and calculus levels have also been demonstrated in asthmatic children.[19] This may be exacerbated by chronic mouth breathing secondary to sinupulmonary disease.

CANDIDA

The long-term use of ICS is strongly associated with increased incidence of oral candidal infection, in particular, pseudomembranous candidosis.[34] This presents as white, plaque-like lesions on the oral mucosal surface, which can be wiped off to reveal an erythematous mucosa. The soft palate and buccal mucosa are commonly affected. Only 10–20% of inhaled corticosteroids are delivered to the lower respiratory tract. The remainder is deposited in the oropharynx. Asthmatic patients have increased incidence of oral candidal infection, which can be attributed to the generalised immunosuppressive and anti-inflammatory effects of the corticosteroids. In addition, many dry powdered inhalers contain lactose monohydrate. The elevated oral glucose levels provide an encouraging environment for candidal adhesion and proliferation in the oral cavity, thus elevating the risk of mucosal infection in the asthmatic patient. As mentioned previously, the use of beta-2 agonists reduces salivary flow, which may further predispose the asthmatic patient to candidal infection.[5]

The risk of oral infection can be minimised by a number of simple methods. It would be prudent to advise the patient to rinse with water or to brush teeth following administration of the inhalers to reduce its topical effects on the mucosa. Increasing salivary flow though chewing sugar-free gum, along with the use of antimicrobial mouthwashes may also reduce the risk of candidal colonisation. Treatment involves the prescription of oral fluconazole or alternatively Nystatin Oral Suspension.[35]

ASTHMA IN THE DENTAL SETTING

It is widely appreciated that fluoride varnish application may be contra-indicated in children with severe asthma (usually those requiring hospitalisation). The reason for this is the possible reaction to the ingredient colophony. Colophony is used in certain fluoride varnish preparations as a natural resin to help the varnish adhere to the tooth surface and is known to cause sensitivity reactions.[36] Clinicians should be aware that there are many different fluoride varnishes on the market, some of which may not contain colophony. However, many are not licensed for caries prevention and this should be taken into consideration by the prescriber.[37]

Anxiety in the dental environment is a common trigger for acute asthma attacks[38] and, as such, clinicians should adhere to stress reduction protocols to avoid such an attack from occurring. Sedation may be indicated, but judicious case selection is necessary in the asthmatic patient. The use of nitrous oxide inhalation sedation in patients with mild-to-moderate asthma is thought to prevent acute symptoms.[39] However, it is advisable to obtain a medical consultation in patients with severe asthma as nitrous oxide has the potential to cause airway irritation, which may exacerbate an attack. A 2003 study by Kil *et al.* found that sedation with midazolam had no adverse effects on paediatric asthmatic patients,[40] a result which confirmed the findings of Fraone *et al.* in 1999.[41] Although no adverse effects were reported, it must be remembered that midazolam is a sedative drug, which may cause respiratory depression, and caution is required when sedating the asthmatic patient.

Vigilance should be exercised when delivering local anaesthetic to dental patients with asthma. This is due to the presence of sodium metabisulphate, which is found in local anaesthetic solution containing vasoconstrictors and is used to prevent the breakdown of the vasoconstrictor. Studies have suggested that this agent may induce hypersensitivity reactions, thereby precipitating an asthma attack.[42,43] There is limited data on the incidence of this problem and, indeed, it is thought that the reaction is not a common one, even in sulphite-sensitive patients. This is due to the fact that dental anesthetics only contain a small amount of metabisulfate. Studies indicate that around 96% of asthmatics are not actually sensitive to sulfites; and those who are, are usually severe, steroid-dependent asthmatics.[44] Although many articles have advised the avoidance of these sulphite-containing solutions, equally the use of local anaesthetics with vasoconstrictors has been used safely in asthmatic patients.[45] Therefore, it is unclear what the true recommendation should be. If a patient has a history of previous sensitivity, excise caution and use a local anesthetic without a vasoconstrictor.

MANAGEMENT CONSIDERATIONS FOR THE ASTHMATIC PATIENT

There are numerous management considerations for the dental treatment of the asthmatic patient (Fig. 1). The main concern in managing asthmatic patients undergoing dental treatment is to avoid an acute attack. This will involve not only care during the procedure, but also care before and after the procedure. As a general dentist, one of the most important things to complete is a full disease history in order to ascertain the severity of the condition and adequacy of asthma control. Note frequency of exacerbations and the date of the last exacerbation. Enquire about frequency of symptoms and any exercise limitation. If the patient is aware of their peak expiratory flow (PEF) or forced expiratory volume in 1 second (FEV1), then record this in the notes. Document hospitalisation and medication history. Record any common triggers (Fig. 2). If the patient is taking a high dose of oral corticosteroids then there may be a risk of adrenal insufficiency and it would be prudent to check with the general medical practitioner whether steroid cover is required for prolonged or stressful treatment.[46] During treatment, care should be exercised to take measures to prevent exacerbation. Aerosols from ultrasonic hand-pieces as well as tooth enamel

- Educate patients about their increased susceptibility to oral disease.
- Encourage regular dental check-ups with enhanced prevention including the prescription of high-strength fluoride toothpaste and fluoride mouth rinse, if age appropriate.
- Advise mouth rinsing or tooth brushing after inhaler utilisation.
- Advise the use of a spacer device to deliver asthmatic drugs directly to the airway.
- Advise the use of sugar-free chewing gum, particularly after meals to increase salivary flow and buffer the acidic effects of medication.
- Be aware that asthmatic patients tend to mouth breath, have a reduced salivary flow and tend to 'wash away the taste of the medication' all of which results in an increase of fluid intake. Therefore, it is important to ensure that patients understand the implications of frequent intake of sugary drinks and advise the consumption of water only.
- Be aware of the increased risk of dental erosion and make appropriate referrals as necessary.

Fig. 1 Dental management of the asthmatic patient

Know your triggers in the dental surgery

- Anxiety
- Aerosols
- Tooth enamel dust
- Residue from dental materials
- Prolonged supine positioning
- NSAIDs
- Opioid drugs
- Products containing sulphites

Fig. 2 Asthma triggers

dust and dental material residues have been shown to be triggers for asthma, together with prolonged supine positioning.[47-49] The use of a rubber dam in such patients may greatly help to reduce the likelihood of an attack occurring and also ensure protection of the airway.

MANAGEMENT OF THE ACUTE ATTACK

A severe acute asthma attack may present as breathlessness and expiratory wheeze. The child may not manage to complete a sentence or may be too breathless to feed. Respiratory rate may be >40/min (2–5yrs) or >30 (>5 yrs). Heart rate may also be increased at >140/min (2–5 yrs) and >125/min (>5 yrs). In the case of a life threatening attack the patient may present with cyanosis, reduced respiratory effort, reduced heart rate, neurological signs such as confusion, or reduced consciousness or loss of consciousness.

Assess airway, breathing, circulation, disability, and exposure. If the patient is conscious, sit them upright and administer two puffs of a short-acting beta agonist (salbutamol 100 mcg/puff inhaler) and repeat if necessary. If the patient is unable to use the inhaler appropriately, administer the drug using a spacer device. Administer oxygen at 15/L minute. If there is no improvement, or the asthma attack is severe, phone for an ambulance.

If the patient loses consciousness, begin cardio-pulmonary resuscitation.[50]

CONCLUSION

Asthmatic patients are at a greater risk of developing dental diseases and the dental setting contains a multitude of common triggers for asthma attacks. This highlights the importance for dental practitioners to be vigilant and to recognise the correlation between asthma and its associated oral health problems. Early diagnosis, correct intervention and prevention can help minimise the risks of developing dental diseases and reduce the occurrence of acute exacerbations.

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