Acidic bronchial secretion in bronchial asthma.

Dr. Tosaddak Ahmed*

Consultant Chest Physician, King Fahad Hospital, Madinah Medical Center, Kingdom of Saudi Arabia

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Editorial

The sputum of patients suffering from bronchial asthma is characteristically acidic [1], unlike the sputum of patients with other lung diseases such as chronic bronchitis, bronchopneumonia and tuberculosis. It is unclear why the sputum is acidic and where the acid comes from. It most probably arises from the breakdown products of catecholamines (adrenaline and nor-adrenaline) produced by the enzymes monoamine oxidase and catechol-o-methyl transferase in the airways. The activation and excessive activity of these enzymes in the bronchial tree have been linked to the initial stages of asthma. Although status asthmaticus is a very stressful state, blood catecholamine levels do not increase as they do in other stressful conditions [2,3]. However, urinary vanillylmandelic acid levels are elevated in status asthmaticus [4].

Increased breakdown of catecholamines results in decreased sympathetic activity in the airway, eliciting bronchospasms (early asthma reactions). Adequate sympathetic activity in the bronchial tree is essential in maintaining the integrity of the mast cells and eosinophils present in the vicinity. When sympathetic activity decreases, mast cells and eosinophils become unstable and degranulate, releasing histamine and leukotrienes that further exacerbate bronchospasms (delayed asthma reactions). We have previously reported in a case study that inhalation of a monoamine oxidase inhibitor (Phenelzine) elicited a greater and more sustained relief of asthmatic symptoms compared to the salbutamol (Ventolin) inhaler (100 microgram) [5]. In that study, a 15-mg Phenelzine tablet was crushed into fine powder that was then administered by a rotahaler. Local activation of monoamine oxidase and catechol-o-methyl transferase in the bronchial tree produces bronchial asthma, while global activation of these enzymes generates anaphylaxis. It would be worth exploring whether monoamine oxidase inhibitors provide faster and greater relief in anaphylactic reactions than adrenaline. Further studies are required to determine how monoamine oxidase and catechol-o-methyl transferase contribute to bronchial asthma and anaphylaxis.

Acidity in the bronchial tree itself, whether it is gastric acid reaching the airway through reflux and aspiration or sulphur dioxide and nitric oxide from the atmosphere producing sulphuric and nitric acid in the airways, causes bronchoconstriction by inducing reflex bronchospasm through the vagus and increasing sputum viscosity. Neutralisation of airway acidity through nebulisation of near isotonic sodium bicarbonate solution alleviates the symptoms of bronchoconstriction and improves the peak expiratory flow rate (PEFR) [6]. This application has saved the life of patients with severe asthma by liquefying the viscid sputum clogging the small airways [7].

Increased expression of inducible nitric oxide (NO) synthase can reverse bronchoconstriction by releasing NO, which dilates bronchial muscle. This is an intrinsic mechanism that occurs in the bronchial tree to reverse asthmatic bronchospasm. The increased production of NO is reflected in its elevated level in expired air, which is used to diagnose and assess bronchial asthma. It has been observed that 5 mg of sublingual isosorbide dinitrate, which delivers NO to the bronchial muscle, significantly alleviates acute asthma symptoms and improves PEFR (unpublished).

Further studies are required to confirm the roles of monoamine oxidase and catechol-o-methyl transferase in the initial stages of bronchial asthma. If these enzymes are shown to have a significant link to asthma, inhibitors against them could be more potent than other bronchodilators.

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***Correspondence to:**

Dr. Tosaddak Ahmed
Consultant Chest Physician
King Fahad Hospital
Madinah Medical Center
Kingdom of Saudi Arabia
Tel: +966 12 660 6111
E-mail: tosaddak1@hotmail.com