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REVIEW

Allergic respiratory diseases in the elderly

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Summary
In industrialized countries there has been a significant increase in life expectancy, but chronic diseases are still important causes of death and disability in the elderly. Individuals over 65 years of age have a decrease in organic functions and lungs can lose more than 40% of their capacity.

Although asthma and allergic rhinitis are more common in young people their prevalence in the elderly is increasing and the mortality reported in these patients is high.

Asthmatic airways show an accumulation of activated eosinophils and lymphocytes determining structural changes of the bronchi. Local allergic inflammation, changes in T cell phenotypes and in apoptosis contribute to systemic inflammation. An increased risk of respiratory infections and neoplasic diseases has been recognized.

These patients have increased susceptibility to atherosclerosis and cardiovascular diseases.

Metabolic diseases are associated with an impairment of lung function and with systemic inflammation. Summing up older asthmatic patients have an increased risk to premature disability and death.

A proper therapeutic approach to asthma can minimize this evolution. To identify the triggers is an important goal that allows reducing medication needs. Corticosteroids dampen allergic inflammation; therefore, they are the first choice in the treatment of patients with persistent asthma and rhinitis. Second-generation H1 receptor antagonists have reduced side effects and can be used if necessary.

The elderly may have difficult access to health care. They should be educated about their disease and receive a written treatment plan. This information improves the quality of life, socialization and disease outcome in older people.

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Contents

Successful ageing and disease

In industrialized countries the significant increase in life expectancy and the declining fertility rates are shifting the age distribution of populations towards the older age groups. Citizens over the age of 65 years account for more than 20% of the European population. The increase in life expectancy is partially due to substantial elimination of infectious diseases through a better hygiene and a widespread use of antibiotics and vaccines. Chronic diseases such as cardiovascular diseases, stroke and cancer still present unmet needs and are the main causes of death in the elderly. Several additional chronic disorders, nutritional disorders and stress contribute to the severity and to the high mortality of the reported diseases. To find out the right answers for health needs, autonomy and successful ageing it is necessary to control the multiple chronic conditions of the elderly.

Ageing is not a disease and this fact is central to understanding the fundamental physiologic changes that develop with age. Failure to distinguish between research on ageing and research on age-associated diseases has been the source of many misunderstandings.[1] In the minds of many biomedical scientists, no one suffers or dies from ageing. People suffer and die from the diseases associated with the ageing process. Death is a result of the inevitable increase in systemic molecular distress that a long living incurs, which enhances vulnerability to severe diseases. The human ageing process affects multiple organs and tissues and involves the progressive deterioration of all body functions.[2] Ageing is not only a programmed process governed directly by genes, but also a stochastic process that occurs after reproductive maturation, which results from the decreased capacity to maintain molecular homeostasis (Fig. 1).

Free radicals and oxidants, commonly called reactive oxygen species (ROS), are highly reactive molecules that can damage all sorts of cellular components inducing disease. ROS can be originated from exogenous sources and from several intracellular sources throughout chronic inflammatory processes. Since oxidative damage of many cell types accumulate with age, the free radical theory of ageing argues that ageing results from the damage generated by ROS. To protect against oxidation there are many different types of antioxidants, such as Vitamins C and E as well as enzymes such as superoxide dismutase (SOD), catalase and glutathione peroxidase.[3] Briefly, antioxidant enzymes are capable of degrading ROS into inert compounds through several chemical reactions. Some enzymes are also involved in the repair of oxidative damaged biomolecules. Methionine sulfoxide reductase A catalyzes the restore of protein-bound methionine residues to methionine repairing the damaged proteins oxidized by ROS.

The simple existence of enzymes to prevent and repair damage by ROS is a strong indicator that ROS are dangerous molecules with an important biological role in disease.[4] The inefficiency in the response of the antioxidant system can result in an increase of oxidative stress, leading to oxidative damage of biomolecules such as DNA, proteins and lipids.

Ageing of respiratory system

Individuals over 65 years of age even with a healthy life style have a decrease in several organic functions. The lungs can lose more than 40% of their capacity over time.[5] Physiologic changes of the lungs due to ageing are characterized by airspace enlargement without alveolar destruction, decrease in gas exchange surface and loss of supporting tissue in the peripheral airways. These changes result in decreased static elastic recoil of the lung, increased residual volume and increased functional residual capacity. Compliance of the chest wall diminishes and the work of breathing increases. As people become older, the respiratory muscles tend to be become weaker and this change is directly correlated with individual nutritional status. The decrease in expiratory flow rates produces a characteristic alteration in the flow-volume curve typical of small airways' disease. The ventilation—perfusion ratio heterogeneity increases as a consequence of premature closing of the distal airways. Cough, that also helps to clear the lungs, tends to be less effective. Nevertheless, in healthy individuals, the respiratory system is capable of keeping an adequate gas exchange during the entire lifespan, only with a slight decrease in arterial oxygen tension.

With ageing there are alterations in the pulmonary performance and in the perception of breathing associated to airway constriction as a consequence of a decline in the sensitivity of the lung receptors.[6]

The decreased sensitivity of respiratory centers to hypoxia can affect the response to airway obstruction. In

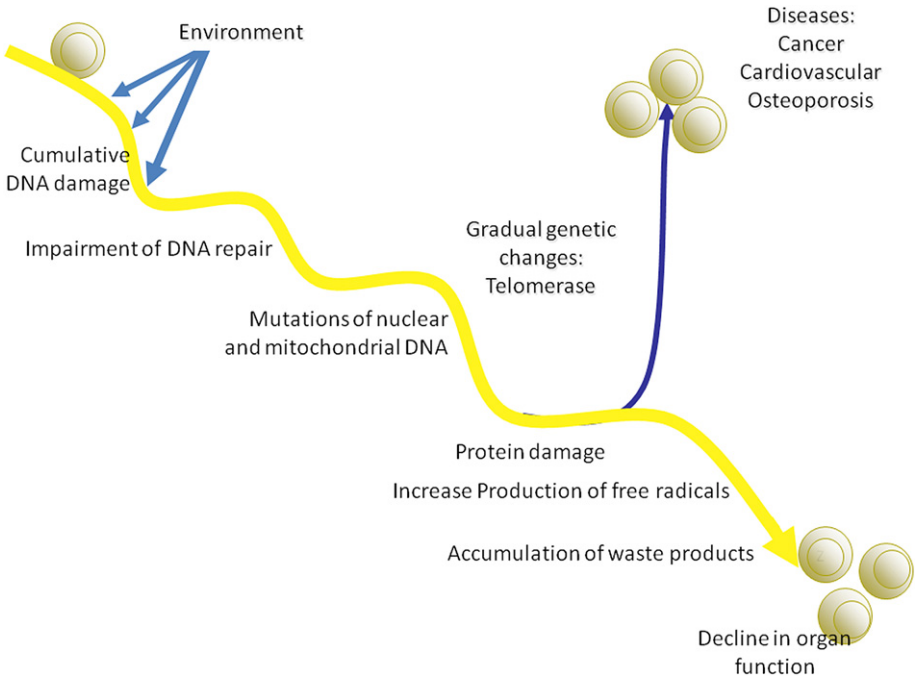

Figure 1 Molecular and cellular mechanisms associated with aging.

addition, decreased perception of dyspnoea may result in lower awareness of respiratory diseases and delay in its diagnosis.[7]

Ageing and respiratory allergy

Although asthma and allergic rhinitis are more frequent in young people, they affect individuals of all ages. Both asthma and rhinitis have a severe impact on patients' quality of life in different domains.

Asthma is a chronic inflammatory disorder of the airways, characterized by a widespread but variable bronchial obstruction and by hyper-responsiveness to several triggers. Allergic rhinitis (AR) is a disorder of the upper airways resulting from IgE-mediated inflammation of the nose upon contact of the nasal mucosa with allergens. This disease, also called allergic rhinosinusitis since histopathologic changes also affect the paranasal sinuses, typically presents with sneezing, nasal pruritus and obstruction. It is often associated with allergic conjunctivitis. Approximately 75% of patients with asthma also have rhinosinusitis, which in turn is a risk factor for asthma. Both diseases share environmental and genetic risk factors. Treatment of upper airway disease can improve asthma symptoms and decrease lower airway hyper-responsiveness.[8]

The prevalence of respiratory allergic diseases in the elderly is increasing and mortality rates are highest among patients over 65 years old with asthma.[9]

The evolution of asthma and the limitation to the airway flow depend on the additional effect of localized inflammatory process, airway remodeling and smooth muscle contraction.[10] Although dyspnoea, chest tightness, cough, and wheezing that characterize asthma in young people are also present in the elderly, an accurate differential diagnosis with chronic obstructive pulmonary disease, congestive heart failure, ischemic heart disease, gastroesophageal reflux, pulmonary embolia, recurrent aspiration, lung tumors, and laryngeal dysfunction should be considered. Drug intake should also be carefully evaluated.

Most of the elderly patients have moderate asthma despite long disease evolution and only about 20% of the patients have severe asthma. Asthmatic patients tend to develop a progressive decline in pulmonary function dependent on age and asthma's duration. However, asthmatics may preserve normal lung function. The poorer prognosis and higher death rates of asthma in older patients are probably associated with chronic systemic inflammation and recurrent exacerbations characteristic of the disease.[11,12] More than half of elderly patients with rhinitis have persistent moderate-severe rhinitis.[13]

Some studies suggest that sensitization to indoor allergens in the elderly may have a more relevant implication in asthma than previously thought. The majority of elderly allergic patients have specific IgE antibodies to indoor allergens and only some of them present positive responses to outdoor allergens in agreement with their higher exposure to indoor allergens. In fact, older patients tend to spend most of their time indoors. Nearly all subjects have positive responses to house dust mite allergen extracts while sensitization to pets is less common.[14,15] The outdoor allergens to which subjects are more often sensitized to are grass pollen in Europe and ragweed in United States of America. Most of these allergic patients suffer exacerbation of their respiratory symptoms during spring or late summer and fall according to their living area.[16,17]

Immuno-inflammatory changes in airways

In allergic respiratory diseases, airways' and systemic inflammation are triggered by repeated contact with

airborne allergens. External antigens bind and cross-link multiple IgE antibodies that are bound to mast cells or basophils. Released mediators promote the recruitment and activation of eosinophils and lymphocytes and amplify inflammatory response and symptoms. The significant relationship established between IgE values or allergic reactivity to common allergens and duration of disease in people older than 65 years of age reinforces the concept that respiratory allergy is present in the elderly.[18] Allergens are transported by airborne particles reaching mucosal surfaces of the airways through inhalation. Upon initial exposure to an allergen, specific Th2 cells are activated and they stimulate B cells to secrete specific IgE. Cross-reactivity between different allergens allows the repeated stimulation and prolongs the lifetime of activated memory cells. Furthermore as allergy is a chronic condition, allergic patients have an increased risk for developing new sensitizations. Total serum IgE concentration can stay within normal range and none relationship between lgE and age or age of onset of asthma has been established.[19,20]

The epithelial barrier is formed by the apical junction of two adjacent ciliated cells called tight junctions (TJ). The TJ control the intercellular transport of inhaled particles and the flow of cell molecules occupying the intercellular space within the epithelium. They are complex structures composed of trans-membrane proteins and receptors such as ocludins, claudins, zonula occludens proteins 1−3 (ZO 1−3), E-cadherin and junctional adhesion molecules (JAM). The intercellular adhesion is due to a structure known as the adherence junction composed of the JAM and E-cadherin, which is connected to E-cadherin of adjacent cells and fixed to the cytoskeleton through protein molecules called catenin. One of 3 the ways to the catenina, the βcateninas, also participates in signaling internal chemical cell systems. These molecules also facilitate communication between adjacent cells.[21,22]

The most relevant allergens such as Der p 1 major allergen, of *Dermatophagoides pteronyssinus*, are able to modify the barrier function of the epithelium and activate both the airway epithelial cells and cells in the immunoinflammatory system. Der p 1 has both cysteine and serine protease activity that act enzymatically breaking the links of occludin, claudins and zonula occludens (ZO) proteins 1−3, thus promoting access to the intraepithelial dendritic cells.

Epithelial cells express poliovirus receptor-related protein (PRR), toll like receptors (TLRs) and protease-activated receptors (PARs), which recognize bacterial agents and allergens. The microorganisms present small molecular sequences designated (pathogen-associated molecular patterns, or PAMPs) that are recognized by TLR and the PRRS. The lipopolysaccaride (LPS) bacteria are considered the prototype of PAMPs.

Recently, a new class of receptors for the recognition of cytosol proteins nucleotide-binding oligomerization domain (NOD) was identified, and recognized their involvement in the intracellular activation after binding to PAR and TLR expressed in epithelial cells. A cascade of intracellular activation is triggered and neutrophils, monocytes and dendritic cells' chemoattractants and cytokines that lead to their maturation are synthesized. These interactions will also influence the intensity of the immune responses and the polarization towards a Th1, Th2, Th17 or T regulatory response.[22,23] Although most of T lymphocytes participating in asthma immune response are of the αβ type there is a small subset of pulmonary γδ T cells involved in allergy and in the defense against pathogens that can decrease during the ageing process.[24] This T cell subset can produce high levels of IFN-γ, which reduces IgE production. These lymphocytes are reduced in elderly patients with long lasting asthma and its reduction favors pulmonary infection.[25]

A decrease in cells with a regulatory phenotype such as Treg CD4hiCD25lo or a failure of Treg cells to give a positive response after contact with the allergen has been found in patients with asthma and other allergic diseases.[26,27] On the contrary, the elderly population seems to have an increase in these phenotypes with the purpose of controlling the quiescent inflammation associated to the ageing process.[28] These biological deviations have a negative impact in clinical outcome of asthma in the elderly.

In asthmatic patients, resistance of different inflammatory cells to initiate apoptosis has been observed, which contributes to maintain ongoing cell activation in the disease.[29] Apoptosis is a classical way of depurating dysfunctional cells and helps to keep tissue homeostasis. Elevated levels of CD95 and higher sensitivity to apoptosis induction seem to be a general feature of all cells in elderly. Elderly asthmatic patients present reduced values of CD95 and reduced susceptibility to start the apoptosis process.[30] This change can facilitate the development of neoplasic diseases.

Many of the attacks to the airways are mediated by the action of ROS. This cell damage may also be implicated in other responses such as the induction of cell death, apoptosis, or neoplastic transformation.

The epithelia damaged and the infiltrated inflammatory cells lead to the generation of growth factors that interact with the mesenchyme promoting airway remodeling and a chronic and persistent inflammatory response.

Asthmatic airway walls present an accumulation of activated eosinophils, lymphocytes, mast cells, macrophages, dendritic cells and myofibroblasts that together determine the bronchial structural changes.[31] Airway remodeling with hypertrophy and hyperplasia of smooth muscle, thickening of the reticular basement membrane and modifications on the airway blood vessels can attenuate the load on airway smooth muscle that usually is necessary during the respiratory circle to overcome the lung elastic recoil force. On the other hand, the thickening of the internal muscle wall can intensify airway smooth muscle shortening and allows it to replace the scarce elastic forces provided by lung recoil.[32] In spite of the apparent positive effect of airway remodeling in bronchi functionality of elderly patients, these changes that are linked to eosinophilic, lymphocytic and neutrophilic inflammation should obviously be avoided and the tissue integrity restored as much as possible.[33,34]

The inflammatory changes present in allergic rhinitis are similar to those reported in asthma. The raise of total IgE tends to be tapered and mast cells, eosinophils and neutrophils have a primordial role in disease pathogeny and evolution (Fig. 2).

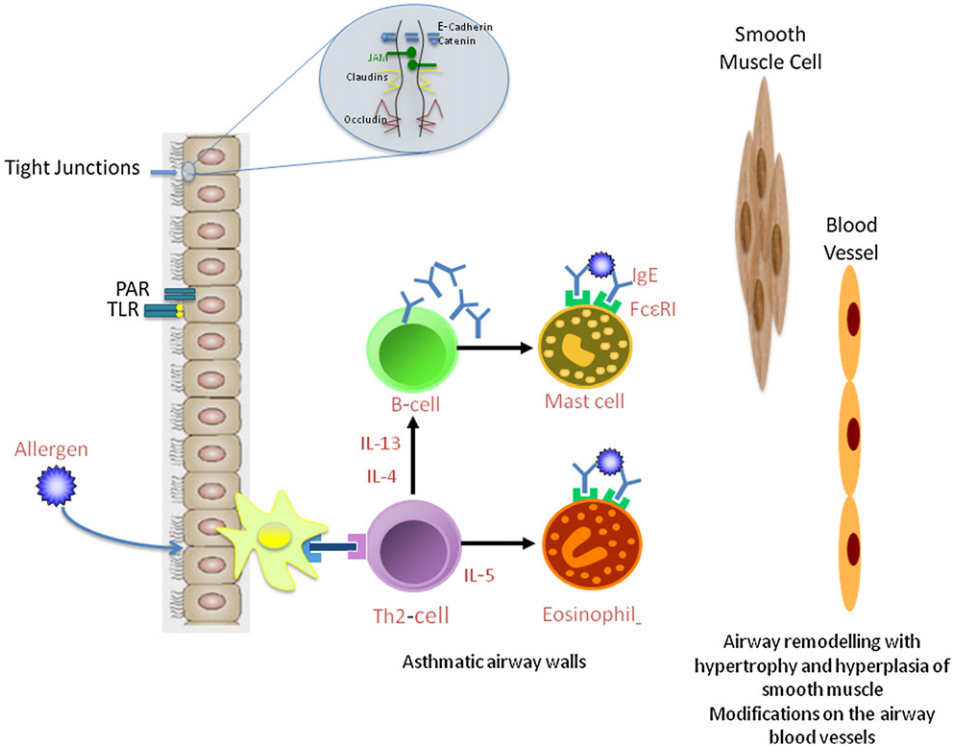

Figure 2 Immuno-inflammatory changes in airways.

Elderly asthma and co-morbidity

The pathophysiology of allergic respiratory diseases is multi-factorial and inflammatory mediators such as histamine, leukotrienes, prostaglandins, and cytokines are released in response to allergic and non-allergic triggers. These mediators enter the peripheral blood and a systemic inflammation becomes established. This sequence of phenomena may increase the risk of cerebrovascular thrombotic events. Allergic rhinitis and exposure of the upper respiratory track to inflammatory triggers such as infections, allergens and pollutants have been recognized as an increased risk for stroke and for hospital admissions caused by other vascular pathologies. Patients using antihistamines can be even at higher risk.[35—37]

Asthmatics have an increased susceptibility to atherosclerosis, which is probably connected with inflammatory pathways inherent to both diseases. Cysteinyl leukotrienes are potent inflammatory mediators implicated in the pathogenesis of asthma and atherosclerosis.[38] An association between carotid intima-media thickness and adult-onset asthma in women has been reported.[39] Women with late-onset of asthma have an increase incident of stroke and coronary heart disease. Cyclic severity of asthma in women is linked to changes in estrogens levels. Usually uncontrolled asthma increases after puberty and after menopause suggesting a hormonal modulation in the disease severity and probably also in inflammatory cytokine and leukotriene activities. Asthma is considered a risk factor for the vascular disorders mentioned, independent of smoking, body mass index, and physical activity.[40,41] Asthma seems to increase atherosclerosis and both are associated with cardiovascular disease.[42,43]

Metabolic diseases such as diabetes can modify asthma evolution. It has been observed that hyperglycemias are associated with impaired lung function, and both hyperglycemias and asthma are associated with systemic inflammation.[44] A diet rich in fruits and vegetables is associated with a decreased risk of newly diagnosed asthma[45] and COPD.[46] The anti-inflammatory effects of Ω-3 fatty acids can modulate inflammatory activity and reduce the risk of asthma and COPD.[47,48] A study directed to obstructive respiratory diseases and wheezing suggests that Ω-3 fatty acid intake does not have the expected protective effect on lung function or disease symptoms, but confirms that Ω-6 acids can have adverse effects on lung function.[49] Obesity is a metabolic or nutritional disturbance, which is also considered a predisposing factor to have asthma, mainly in women. Dyspnoea is a result of bronchoconstriction and dynamic hyperinflation, which is amplified in obese individuals. Symptoms and respiratory function tests may be difficult to assess in obese asthmatic patients because pulmonary dynamic is affected by a reduction in chest wall compliance and by changes in the airway resistance. The recognized reduction in static lung volumes, particularly in functional residual capacity (FRC) and total lung capacity (TLC) can be associated to a reduction also in forced vital capacity (FVC) in severe obesity. These effects of obesity on respiratory function may work in the opposite direction from asthma, and can justify normal lung volume measurements in obese individuals with asthma. Response to bronchi provocation tests in obese individuals, shows an increase in FRC and airtrapping and a reduction in inspiratory capacity (IC). Usually during bronchoconstriction, there is an increase in the activity of the inspiratory muscles to shorten the expiration and to maintain

ventilation. This reduced activity may be caused by the effect of fat on chest wall recoil.[50–54] The increased risk for asthma in obese is not a feature of elderly asthma but affects all age groups.

Total Antioxidant Status (TAS) and SOD values are usually higher in healthy individuals than in asthmatic patients.[55] The reduction of the anti-oxidative capacity observed in patients with long lasting asthma may have a negative influence in other clinical conditions occurring in elderly.

Several studies have pointed out an association between lung cancer and other pulmonary diseases such as bronchial asthma, chronic bronchitis, emphysema and lung tuberculosis after adjustment for smoking habits. Increased risk of developing lung cancer has been reported in individuals with cough, dyspnoea and effort dyspnoea in both genders. The risk increases with the number of symptoms reported, mainly for non-small cell lung cancer.

It is anticipated that chronic inflammatory process and reduced apoptosis may stimulate local cell-proliferation and growth, favoring malignant pulmonary diseases. Trying to modulate pulmonary inflammation may help to prevent later development of lung cancer.[56–60]

Pneumonia and influenza infection have long been considered one of the most common health problems of old people. The infection causes inflammation, deterioration of lung function and reduction of oxygen blood transfer increasing breathing effort. Older people have weakened defenses against infection. Cough is not as effective as in younger people. Elderly populations whose lungs have been damaged by chronic obstructive pulmonary disease develop repeated infections and severe dyspnoea attacks.

Therapeutic and ageing conditionings

Older patients with multiple morbidities may have an aversion to taking multiple drugs, mainly those that should be taken regularly. They usually prefer drugs that can be adjusted to their symptoms. The cost of the medication may also be a limitation for adhesion to treatment. Older people may also assess differently their symptoms of asthma since they consider dyspnoea a less bothersome symptom than younger patients.[61] Drugs without recognized clinical benefit should be withdrawn.

The initial approach to treat allergic diseases starts with an accurate identification of triggers to avoid the offender antigens when possible and to reduce medication needs. Patients should also be advised to avoid non-allergic factors which may induce their respiratory symptoms, including irritants as cigarette smoke and household aerosols. Patient's medication list should be analyzed to detect any medications that can exacerbate asthma, such as beta-blocker eye drops and anti-hypertensive therapy.

Controller medications are indicated if rescue medications are often required (more than twice a week).

Corticosteroids dampen the inflammatory reaction characteristic of allergy; therefore they are the first choice treatment for long-term in patients with persistent asthma and persistent rhinitis. Untreated chronic airway inflammation may produce structural changes in the airways and fixed obstruction. This is pertinent, because most of the elderly patients that are hospitalized for asthma were not using any controller medication in the year preceding the asthma exacerbation.[62,63]

Inhaled corticosteroids can produce good effects at low doses having few systemic adverse effects and being safe in the elderly. Low doses of inhaled steroids demonstrate significant change in airway mucosal inflammation. To reduce the basement membrane thickness in asthmatic patients, the use of higher doses of inhaled steroids is determinant but this approach is more important in the initial phases of the disease.[64] However, cognitive and sensory impairment compounds and also co-morbidities such as rheumatoid diseases can affect self-handling of inhaler devices. Dry powder inhalers require appropriate inspiratory flow, which excludes their administration to some patients. The most convenient inhaler device for an old person is a spacer with a metered dose inhaler. The compliance to the different types of aerosol should be considered in elderly patients. Their use can be associated to local adverse reactions such as oropharyngeal candidiasis and hoarseness, which can be prevented by using a spacer or by rinsing the mouth after use. If patients receive very high doses of ICS, they have potential for systemic absorption, with suppression of hypothalamus, pituitary and adrenal function and side effects similar to those receiving low doses of oral corticosteroids. As a consequence they can also develop cataracts, glaucoma and osteoporosis. Oral steroids can be used in more severe conditions for short periods of time.

Nasal corticosteroids are the first line of medical therapy for allergic rhinitis since they rarely produce serious side effects and they are effective in all forms of rhinitis.

Elderly patients treated with high doses of corticosteroids should be regularly submitted to bone density measurements to evaluate osteoporosis and the risk of bone fractures. Exercise should be encouraged and supplements of oral calcium with vitamin D should be considered. Ophthalmologic examination to check vision, slit lamp exams to look for cataract and intraocular pressure testing are also recommended. To follow blood glucose levels are recommended for known diabetics and blood pressure measurement should be performed in each visit.

To reduce the dose of ICS or the need for oral steroids, steroid-sparing medications such as leukotriene receptor antagonists and long-acting beta-agonists may be added.

Oral leukotriene antagonists are CysLT1 receptor antagonists that provide rapid improvement of respiratory function. They are safe but liver damage has been observed in a little number of cases.

The β2 agonists are drugs that relax smooth muscle and promote the removal of airways secretions by ciliar movement. The formulations of long-term β2 agonists are used as controller medications in asthma. While long-acting beta-agonists currently available are safe, they should be used with caution in patients with cardiac disease. The density of β2-receptors decreases with age and consequently the β2 agonists may be less effective in elderly patients with asthma.

Short-acting β2 agonists are relatively safe in the elderly if used as rescue medication to revert exacerbations. Although systemic absorption can produce tachycardia, palpitations and tremor they are less common with inhaled formulations. Both short-acting and long-acting β2 agonists

should be used with care in patients with hyperthyroidism and diabetes. Overdose may cause hypokalemia and arrhythmia.

An alternative rescue medication to short-acting β2 agonist is ipratropium bromide. Anticholinergic agents are useful alternative, particularly in the case of partial relaxant effects and when an adverse effect upon administration of β2-mimetics occurs.[65]

Allergen immunotherapy should be reserved for patients whose medical approach of allergic rhinosinusitis or asthma fails. Although effective in younger patients, has a questionable efficacy in elderly and is not without risk. Adverse reactions range from local irritation till anaphylaxis and epinephrine therapy for rescue may be ineffective or associated with acute hypertension. Immunotherapy is also contraindicated in patients with persistent severe asthma where adverse respiratory reactions may be difficult to treat.

Although most of the effects of histamine in allergic disease occur through histamine-1 (H1) receptors, hypotension, tachycardia, flushing and headache occur through both the H1 and H2 vascular receptors, whereas cutaneous itch and nasal congestion may occur through the H1 and H3 receptors. In addition to its role in the early allergic response to antigen, histamine acts as a stimulatory signal for the production of cytokines and the expression of cell-adhesion molecules and class II antigens, thereby contributing to the late allergic response. Histamine exerts other important immunomodulatory effects through H1, H2, H3, and H4 receptors. The four major types of histamine receptor differ in their expression, signal transduction, and function. H1 and H2 receptors are widely expressed, in contrast to H3 and H4 receptors. Antihistamines block the effects of histamine, the main mediator of the immune system in a type I allergic reaction and is recommended in all forms of rhinitis and allergic diseases. They are associated with adverse effects such as drowsiness or malaise so their use in elderly patients should be carefully evaluated. The first generation of H1 receptor antagonists should be avoided as they lack specificity for the H1 receptor and produce a variety of side effects in elderly, including anxiety, confusion, sedation, disequilibrium, postural hypotension, constipation and urinary retention. The second generation of H1 receptor antagonists has reduced side effects, low cross blood brain barrier and greater specificity for its receptor.

All the first-generation H1-antihistamines and some second-generation H1-antihistamines such as desloratadine and loratadine are metabolized by the system cytochrome P450 hepatic. Cetirizine is excreted largely unchanged in the urine, and fexofenadine is excreted largely unchanged in the feces. Interactions that result in changes in plasma concentrations can diminish or increase adverse effects. Interactions may be more likely to be associated with first-generation H1-antihistamines than second-generation H1-antihistamines, which have a wider therapeutic index.

Theophylline has a narrow therapeutic window and is not considered the first choice medication in the elderly. Nevertheless theophilin is less expensive than other therapeutic drugs for asthma and in low blood concentration have anti-inflammmatory effects.[66] Adenosine is a biological mediator with the capacity of producing pro inflammatory effects in part through A(2B) adenosine receptors response of mast cells, epithelial cells and smooth muscle cells. Theophilin can act as both bronchodilator and immunomodulator depending on the serum concentration achieved. The antagonism of adenosine receptors occurs in lower dose than that required for inhibition of phosphodiesterases. Accordingly theophyline is safe if properly used and should be reconsidered in asthma treatment in all aged groups.[67,68]

Influenza vaccine should be administered annually to prevent old patients from getting influenza and is especially recommended if they have a chronic medical problem, such as asthma. Effectiveness of routine use of pneumococcal vaccine in people with asthma is still controversial even in elderly.[69]

Other therapeutic alternatives such as thromboxane A2 antagonists or even cytokine inhibitors should be considered in particular cases.

Elderly usually have difficulties to access to health services. The asthmatic patients should be educated about their disease and receive a written treatment plan. This plan is a guide with information about controller and reliever medication and should also be explained to family members. Patients should be able to recognize an asthma exacerbation and what to do to reverse it. This information improves the quality of life, socialization and hobbies throughout the ageing process.

Conclusion

It is important to recognize that allergic respiratory diseases often start during childhood but they can last for life. New sensitizations may occur at any age and elderly people have a higher risk of having multiple allergy syndrome. The local inflammatory disabilities can produce structural changes in the airways and when extended systemic immune-inflammatory deviations can be observed. An aggressive therapeutic approach should be implemented in early states of the disease to minimize these changes. In elderly a more conservative therapy should be selected, but the control of inflammation has to be assured. It is important to assure respiratory function and also to reduce asthma-associated respiratory diseases.

Conflict of interest

The authors have no conflict of interest.

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