Pulmonary Pharmacology & Therapeutics 35 (2015) 1–7

Contents lists available at ScienceDirect

Pulmonary Pharmacology & Therapeutics

journal homepage: www.elsevier.com/locate/ypupt

Review

Therapeutic approaches of uncomplicated arterial hypertension in patients with COPD

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ARTICLE INFO

Article history:
Received 28 July 2015
Received in revised form 3 September 2015
Accepted 6 September 2015
Available online 10 September 2015

Keywords:
Hypertension
Chronic obstructive pulmonary disease
Diuretics
Beta-blockers
Calcium channel blockers
Renin-angiotensin system blockade

ABSTRACT

The concomitant presence of systemic arterial hypertension and chronic obstructive pulmonary disease (COPD) is frequent. Indeed, arterial hypertension is the most common comorbid disease in COPD patients. Since many antihypertensive drugs can act on airway function the treatment of arterial hypertension in COPD patients appears complex. Moreover, in these patients, a combined therapy is required for the adequate control of blood pressure. Currently, available data are inconsistent and not always comparable. Therefore the aim of this review is to analyze how antihypertensive drugs can affect airway function in order to improve the clinical management of hypertensive patients with COPD. Thiazide diuretics and calcium channel blockers appear the first-choice pharmacological treatment for these patients.

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Contents

1. Introduction

Chronic obstructive pulmonary disease (COPD) is an important public health issue and is expected to became the third leading cause of death worldwide by 2020 [1–4]. The incidence of COPD increases markedly with advancing age, and is greater in men [2,3]. In addition, Arterial Hypertension (AH) and COPD often are present in the same patient, so that AH is the most common comorbidity in COPD patients [5]. Indeed, Fumagalli et al. [6] reported that AH was present in over 50% of COPD patients referred to Pneumology Unit in the four major hospitals in Rome. In this study, the authors described a significantly greater prevalence of COPD as compared to the report of World Health Organization (WHO),i.e.35% in high income Countries and 45% in low income countries [7]. Probably, this difference is related to the greater proportion of severe COPD (GOLD class 3 and 4) in the Fumagalli et al. [6] study.

The increased arterial stiffness, observed in COPD patients, may predispose to systemic arterial hypertension and other cardiovascular diseases [8], as shown in Fig. 1. AH treatment and control represent a clinical challenge, particularly in case of concomitant risk factors and comorbidities, such as COPD [9]. The Seventh Report of The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure [10] suggests that the treatment goal for individuals with hypertension is < 140/90 mmHg. More than two-thirds of hypertensive subjects require more than one antihypertensive agent to achieve adequate hypertensive control

Abbreviation: COPD, chronic obstructive pulmonary disease; WHO, World Health Organization; AH, arterial hypertension; CHF, congestive heart failure; RAS, renin angiotensin system; AngII, angiotensin II; MCO-1, monocyte chemotactic protein-1; IL-6, interleukin-6; TGF-β, transforming growth factor-β; CCB, calcium channel blockers; LVSD, left ventricular systolic dysfunction.

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http://dx.doi.org/10.1016/j.pupt.2015.09.004
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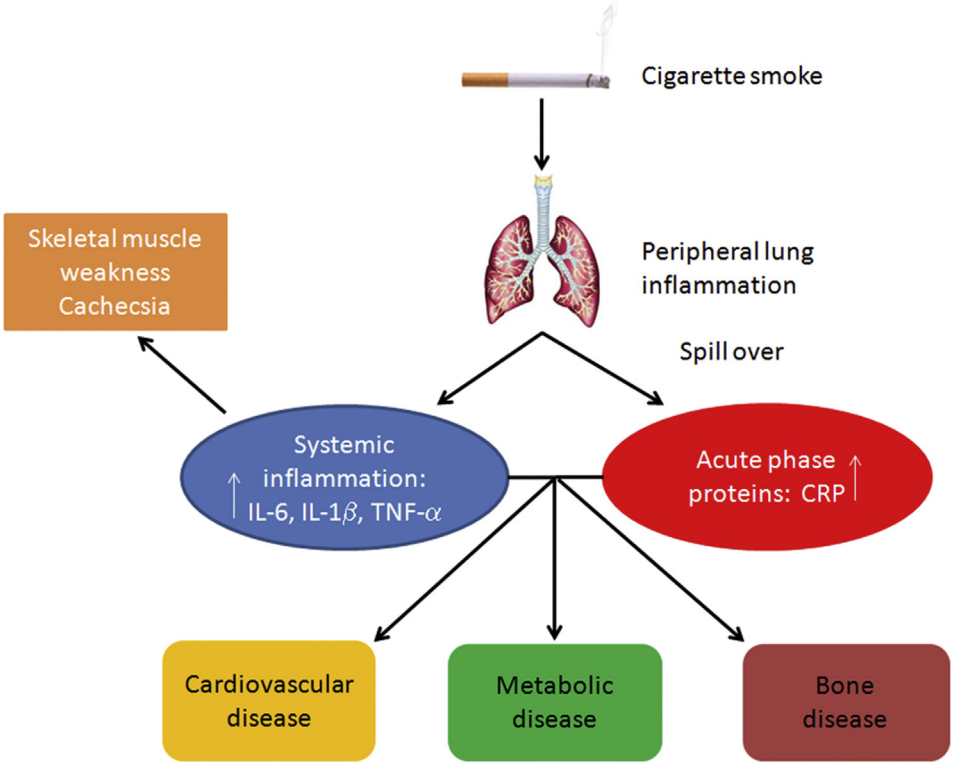

Fig. 1. Pathophysiological mechanism whereby cigarette smoke causes systemic inflammation. IL-6: Interleukin6; IL-1β: Interleukin 1β; TNF-α: tumor necrosis factor α; CPR: C Protein Reactive.

[11–14]. In ALLHAT, 60% of those whose blood pressure was controlled to <140/90 mmHg received two or more antihypertensive agents, and only 30% overall were controlled on one drug [11].

The aim of this review is to analyze how antihypertensive drugs can affect airway function and can contribute to a better clinical management of patients with hypertension and COPD.

2. Diuretics

The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure [10] and the ALLHAT [11] recommend the use of low-dose thiazides for initial pharmacological treatment of most patients with uncomplicated hypertension when the patients do not report contraindications to these drugs [15,16]. In addition, in a recent retrospective study on 7140 patients with hypertension and COPD requiring two antihypertensive drugs, the combination therapy that included a thiazide diuretic was associated with a reduced risk of hospitalization for congestive heart failure (CHF) among patients without a history of CHF. Moreover, in this study the use of a thiazide diuretic in combination therapy was not significantly associated with risk of COPD exacerbations [17].

Thiazide diuretics do not show significant adverse effects on airway function and may be considered the first-choice drug for initial therapy in patients with asthma [18,19]. However, the use of diuretics may interfere with mucus production and cause acid-base and electrolyte abnormalities [20]. An interesting study demonstrated that the treatment with thiazide diuretics augments thehypokalemic and electrocardiographic effects of high-dose inhaled beta-2 receptor agonists such as albuterol. In fact, the latter drives potassium into the cells. Therefore, the arrhythmogenic potential of this interaction may be important in patients with acute exacerbations of COPD, who have concomitant hypoxemia [21].

Hypokalemia from thiazides is dose-dependent; therefore, it is advisable to administer low doses of diuretics to non edematous hypertensive patients with COPD in order to minimize the occurrence of adverse effects.

Although potassium wasting diuretics are the preferred drugs for treating hypertension in patients with COPD, they may worsen carbon dioxide retention in hypoventilating patients and potentiate hypokalemic in those receiving corticosteroids [22].

In conclusion, hypertensive patients with COPD in treatment with potassium wasting diuretics who have chronic respiratory acidosis or are receiving corticosteroids or beta-agonists should undergo close monitoring of electrolyte levels and be considered for therapy with potassium supplement or potassium-sparing agents [22].

3. Renin-angiotensin system blockade: ACE-inhibitors and angiotensin II antagonists

The renin angiotensin system (RAS) may be involved in the pathogenesis of pulmonary and extrapulmonary manifestations that are observed in patients with COPD [23]. In fact, a local RAS is present in many human tissues, including lung and skeletal muscle [24]. Electron microscope studies with microperoxidase labeled antibody against angiotensin-converting enzyme localize the enzyme activity to the luminal surface of the capillary endothelium and associated caveolae in the lungs [25]. Furthermore, Angiotensin II (Ang II) receptors are also expressed in the lungs [26]. The chronic activation of the RAS in human is thought to influence cardiopulmonary diseases [27]. The RAS is therefore potentially involved in the pathogenesis of COPD inducing the production of proinflammatory mediators in the lung [28]; in particular Ang II stimulates the release of interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α) and monocyte chemotactic protein-1 (MCP-1) [29]. Alveolar MCP-1 is able to activate tissue mast cells following acute alveolar hypoxia thus trig-

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