



STUDY THE ANTI-INFLAMMATORY EFFECTS OF TAMSULOSIN BY THE EVALUATION OF INFLAMMATORY CELLS AND LUNG HISTOPATHOLOGY IN AN AIRWAY INFLAMMATION MODEL IN RATS

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Objective: Study the role of tamsulosin on the respiratory inflammation in rats with ovalbumin (OVA) induced airway sensitization by evaluating the inflammatory cells in the broncho-alveolar lavage fluid (BALF) and the lung histopathology. **Materials and methods:** Thirty adult male albino rats were allocated into 5 groups (n=6). Group A – Normal control (NC) fed commercial pellets and water. Group B – (as negative control) – subjected to an airway OVA-sensitization. Group C (as positive control) – treated with oral prednisolone (4.12 mg/kg) plus OVA-sensitization. Group D – treated with oral tamsulosin (35 mcg/kg/d, equivalent to 0.4 mg for a 70 kg human) plus OVA-sensitization. Group E – treated with oral tamsulosin (17.5 mcg/kg/d, equivalent to 0.2 mg tamsulosin for a 70 kg human) plus OVA-sensitization. Inflammatory cells count/ μ l in the BALF was calculated along with histological analysis of the lung tissue. **Results:** Both doses of tamsulosin (35 and 17.5 mcg/kg/d) significantly reduced the total WBC count, eosinophils, and neutrophils. A significant reduction in mononuclear cells was detected after treatment with 35 mcg/kg/d tamsulosin. Also, the histopathological examination revealed that both doses (35 and 17.5 mcg/kg/d) of tamsulosin caused less agglomeration of the inflammatory cells within the lung tissue and clear alveolar sacs. **Conclusion:** the administration of tamsulosin in rats with induced airway sensitization resulted in protection from respiratory inflammatory events.

Keywords: Airway inflammation; Broncho-alveolar lavage fluid; Inflammatory cells count; Ovalbumin; Tamsulosin.

INTRODUCTION

Chronic airway diseases such as asthma and chronic obstructive pulmonary disease (COPD) continue to be a major burden on individuals and healthcare systems across the world. Asthmatic patients are at risk for respiratory complications, bronchospasms, prolonged hospital stays, and increased mortality¹. Symptoms can range from breathing difficulties and wheezing to coughing, which are associated with variable airflow limitation, and can be difficult to treat². In general, asthma is associated with the activation of the immune system, eosinophilic infiltration,

airway hyper-responsiveness, mucus overproduction, and remodeling of the airways by the immune system^{3&4}. Furthermore, COPD has become a major cause of mortality, it was classified as the third leading cause of death worldwide^{5&6}. COPD patients are at risk for infection, lung cancer, and sudden episodes of acute pulmonary embolism^{7&8}. The chronic condition is characterized by irreversible, continuous airflow limitation and a poor quality of life^{9&10}.

Prior studies have confirmed the presence of eosinophilic infiltration in the airways during pulmonary inflammatory disease^{11&12}. Airway inflammation is also caused by the

infiltration and agglomeration of several major inflammatory cells such as neutrophils, monocytes-macrophages, and lymphocytes within the airways and pulmonary tissue¹³. Further, previous investigations revealed extreme infiltration and activation of neutrophils and monocytes in the airways in response to inflammatory disease^{14&5}. Monocytes can develop into macrophages or dendritic cells during the inflammatory process when stimulated by specific pro-inflammatory cytokines¹⁴.

Current anti-inflammatory medications aim to reduce symptoms and avoid exacerbations¹⁶. Corticosteroids are the treatment of choice for the management of airway inflammatory events¹⁷. However, even with current guidelines, 45 to 55% of asthmatic patients continue to experience uncontrolled symptoms and are not able to recover from the disease¹⁸. Drawbacks in the treatment of airway inflammatory disease include the lack of safe and effective disease-modifying therapies. Therefore, finding an alternative medical option should be a primary research goal.

Tamsulosin acts as an antagonist for the alpha-1-A adrenergic receptor (α_{1A} -AR) and it is globally prescribed as a first-line treatment to treat lower urinary tract symptoms (LUTS) caused by benign prostatic hyperplasia (BPH)¹⁹. A previous study has confirmed that tamsulosin can reduce the production of mRNA of numerous inflammatory genes that govern the LUTS²⁰. However, tamsulosin may also attach to α_1 -AR in organs other than the urinary tract. In a previous study that examined tamsulosin distribution, radioactive tamsulosin binding at the α_1 -AR in lung tissue was detected at 10 min following an intravenous injection in rats²¹. Furthermore, the α_1 -AR antagonists were shown to prevent the cytokine-storm syndrome and reduce pneumonia mortality in Coronavirus Disease 2019²². Furthermore, a retrospective analysis of hospitalized patients with acute respiratory distress syndrome found that those who used α_1 -AR antagonists had lower death rates and a decreased chance of needing mechanical ventilation²³. In addition, the use of tamsulosin produced superior results in histological studies which showed a greater restoration of the

hepatic architecture and less fibrosis after inducing hepatic cell damage and inflammation in rats compared to controls^{24&25}.

When used for the treatment of BPH, tamsulosin is safe and may have minimal adverse effects, most notably dizziness, weakness, and nausea²⁶. Furthermore, tamsulosin safety extends to pregnancy as the Food and Drug Administration has classified it under Category B pregnancy risk (described as having no demonstrated risk in animal reproduction studies but insufficient research in pregnant women)²⁷.

The purpose of this study was to evaluate the effectiveness of tamsulosin in treating airway inflammatory disease by suppressing inflammatory cells and preventing the pathological alterations of lung tissue in rats with ovalbumin-induced sensitization.

MATERIALS AND METHODS

Medication, chemicals, and materials used in this work include prednisolone (Wockhardt®, Wrexham, UK), tamsulosin (Astellas Pharma®, Chicago, Illinois, USA), sodium phenobarbital (VERVE®, Turkey) ovalbumin powder (Fisher Scientific Ltd, New Hampshire, UK), aluminum hydroxide (MERCK® Darmstadt, Germany), Normal Saline (N/S) 0.9% (Pioneer, Sulaymaniyah, Iraq) and liquid formaldehyde (37-41%) (S.D. Fine Chem Ltd, Mumbai, India).

Animals

A total of thirty (30) healthy adult male albino rats, 2-3 months old and 150-250 gm in weight. The rats were obtained from the animal house in the College of Veterinary Medicine, University of Basrah and were randomly housed in polypropylene cages at the Animal House at Pharmacy College, University of Basrah. First, they adapted to their environment for 14 days by setting the optimum temperature of 21 ± 4 degC, light-dark photoperiods (12L:12D), and avoiding unnecessary stress. Rats were fed a commercial pellet diet and provided with clear tap water throughout the experiment.

- **Group A** – Normal control (NC) rats were fed commercial rat pellets and water for 14 days.

- **Group B** – (as negative control) – rats were subjected to airway sensitization with the use of OVA.
- **Group C** (as positive control) – rats were orally treated with prednisolone (4.12 mg/kg)²⁸ and subjected to OVA-airway sensitization.
- **Group D** – Rats were orally treated with tamsulosin (35 mcg/kg, equivalent to 0.4 mg for a 70 kg adult patient as shown in (Table-1)) and subjected to OVA -airway sensitization^{20&29}.
- **Group E** – Rats were orally treated with tamsulosin (17.5 mcg/kg, equivalent to 0.2 mg tamsulosin for a 70 kg adult patient as shown in (Table-1) and subjected to OVA-airway sensitization^{29&30}.

- **Second dose** = 0.2 mg/day = 200 mcg
- Average human body weight = 70 kg

The applied model of airway inflammation in rats via OVA-sensitization (in all groups except for group A) was modified by previous researchers^{28&33}, as explained in (Table 2). In the prednisolone and tamsulosin-treated groups, drug doses were given 60 min before exposure to airway sensitization with OVA²⁸. The rats were euthanized using an IP injection of 800 mg/kg sodium phenobarbital³⁴. Broncho-alveolar lavage was performed with 3 mL N/S via a catheter inserted into the trachea. The inflammatory cells in the BALF were counted using an automated hematology analyzer (Sysmex XN-350).

The left lung was ligated, washed with saline, and stored in 10% formaldehyde-filled cups for histopathological analysis. The segments were inspected and photographed using a digital camera attached to a light microscope for amplification at X10 and X40. Inflammatory cells that infiltrated the bronchi and alveoli were examined in a series of H&E-stained lung sections.

Calculation of Prednisolone Dose: Animal does (mg /kg) = 4.12 (mg/kg)²⁸.

In this study, the doses of tamsulosin were chosen based on the effective standard- and low-human doses 0.4 and 0.2 mg, respectively^{31&32}.

Calculation of tamsulosin dose in rats: Dose of tamsulosin HCl in rats = human dose (mcg/kg/day) x 6.17²⁹, as shown in (Table-1).

- **First dose** = 0.4 mg/day = 400 mcg

Table 1: Calculation of Tamsulosin Doses in Rats.

Dose number	Human dose (mg)	Human dose ÷ 70 (mg/kg/d)	Human dose (mcg/kg/d)	Dose in rats (mcg/kg/d)	Dose of 200g rat
Dose 1	0.4	0.0057	5.7	35	7 mcg
Dose 2	0.2	0.0028	2.8	17.5	3.5 mcg

Table 2: The induced airway inflammation model in rats.

Days	Action
1-3	Sensitization with IP injection of (1 mg OVA and 100 mg Aluminum hydroxide in 1 mL N/S), once daily
4-5	No action
6-8	Sensitization with IP injection of (100 mg OVA and 100 mg Aluminum hydroxide in 1 mL N/S), once daily
9-14	Challenging by containing the rats in a glass chamber (30 x 30 x 30 cm) connected to a nebulizer that provides 1% OVA aerosol (1 gm OVA in 100 mL N/S) to be inhaled for 30 minutes each day
15	Euthanizing and sacrificing the rats

IP, intraperitoneal; OVA, ovalbumin; N/S, normal saline 0.9%.

Ethical Approval

Animal experiments were conducted within the ethical framework of the European Union Directive (86/609/EEC) of 24 November 1986 and approved by the local ethical committee.

Statistical analysis

In this study, to represent mean ± SEM, bar graphs were created utilizing Prism, Version 9. The Statistical Package for the Social Sciences (SPSS), Version 20 was used for statistical analysis. The statistical difference was calculated using the one-way analysis of variance (ANOVA) and the unpaired student's t-test. The limit for statistical significance was defined at $P < 0.05$.

RESULTS AND DISCUSSION

Results

Effect of Tamsulosin on Eosinophils Count/ μ l in the BALF of Rats with OVA-Sensitized Airway

After the airway sensitization, the eosinophils count in the negative control group was significantly ($P < 0.05$) higher than that in the NC group (15.16 ± 1.01 versus 3.33 ± 0.76). While in the group treated with prednisolone and both doses of tamsulosin (35 and 17.5 mcg/kg) the eosinophils count was significantly ($P < 0.05$) decreased in comparison to that in the negative control group (7.33 ± 0.71 , 10.00 ± 0.57 and 11.16 ± 1.24 versus 15.16 ± 1.01) respectively, as shown in (Figure 1).

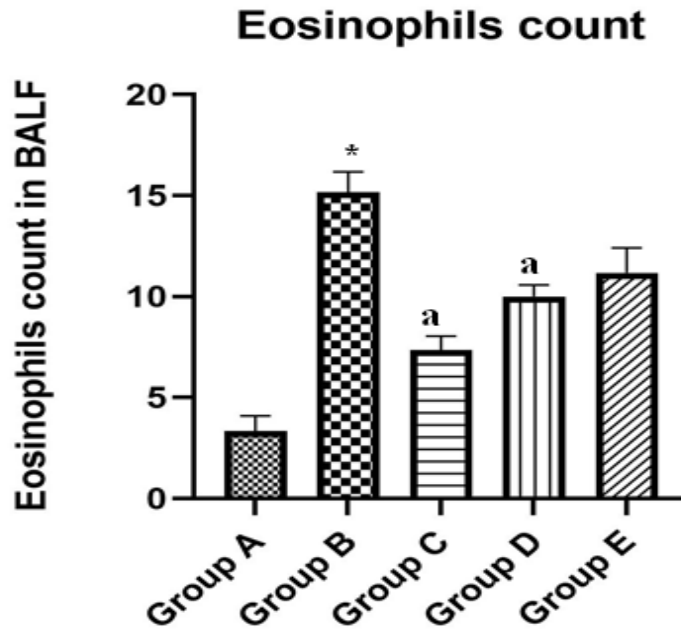


Fig. 1: Effect of tamsulosin on eosinophils in the BALF in rats. OVA-sensitization led to an elevation in eosinophils levels in the BALF, while both doses (35 and 17.5 mcg/kg) of tamsulosin treatment reduced these levels. Group A= normal control; Group B= negative control (OVA-sensitized airway); Group C= positive control (treated with prednisolone); Groups D and E= treated with tamsulosin (35 and 17.5 mcg/kg respectively); *= significant difference ($P < 0.05$) in comparison to group A; a= significant difference ($P < 0.05$) in comparison to group B.

Effect of Tamsulosin on Neutrophil Cells Count/ μ l in the BALF of Rats with OVA-Sensitized Airways

Neutrophil count in the BALF was significantly ($P < 0.05$) higher in the negative control group in comparison to that in the NC group (36.16 ± 7.48 versus 11.66 ± 2.33). Additionally, the neutrophil counts in the OVA-sensitized rats treated with prednisolone and both doses of tamsulosin (35, and 17.5 mcg/kg) were significantly ($P < 0.05$) reduced in comparison to that in the negative control (21 ± 4.56 , 20 ± 3.74 , and 20 ± 4.16 versus 36.16 ± 7.48), respectively. Furthermore, the neutrophils count was significantly ($P > 0.05$) increased in the groups treated with both doses of tamsulosin (35, and 17.5 mcg/kg/d) when compared to that in the NC group (20 ± 3.74 and 20 ± 4.16 versus 11.66 ± 2.33) respectively, as shown in (Figure 2).

Effect of Tamsulosin on Mononuclear Cells (Lymphocytes, Macrophages) Count/ μ l in the BALF of Rats with OVA- Sensitized Airways

The mononuclear cell count in the BALF was significantly ($P < 0.05$) higher in the negative control group in comparison to that in the NC group (33.66 ± 3.63 versus 17.66 ± 3.33). Additionally, the mononuclear cell counts in the OVA-sensitized rats treated with prednisolone and tamsulosin (35 mcg/kg/d) were significantly ($P < 0.05$) reduced in comparison to that in the negative control (19.83 ± 2.78 and 22.50 ± 3.93 versus 33.66 ± 3.63), respectively. Furthermore, treatment with the lower dose of tamsulosin (17.5 mcg/kg) resulted in a reduction in mononuclear cell count when compared to the negative control (24.66 ± 4.78 versus 33.66 ± 3.63), although there was no statistical difference, as illustrated in (Figure 3).

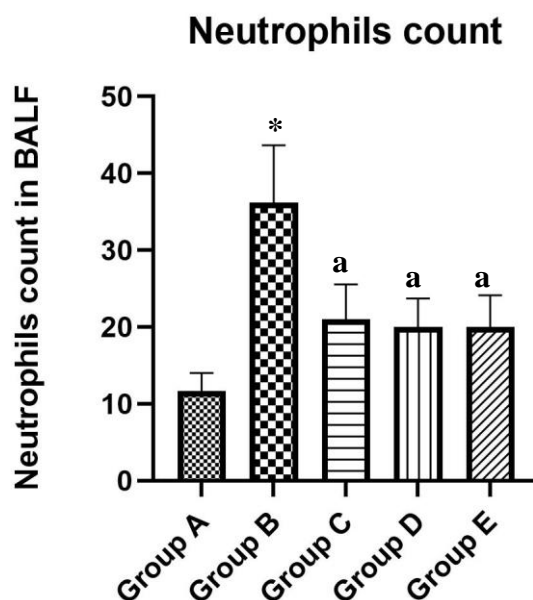


Fig. 2: Effect of tamsulosin on neutrophils in the BALF in rats. OVA-sensitization was associated with the high neutrophil count in rat BALF while tamsulosin treatment in both doses (35 and 17.5 mcg/kg) reduced its expression. Group A=normal control; Group B= negative control (OVA-sensitized airway); Group C= positive control (treated with prednisolone); Groups D and E= treated with tamsulosin (35 and 17.5 mcg/kg respectively); *= significant difference ($P < 0.05$) in comparison to group A; a= significant difference ($P < 0.05$) in comparison to group B.

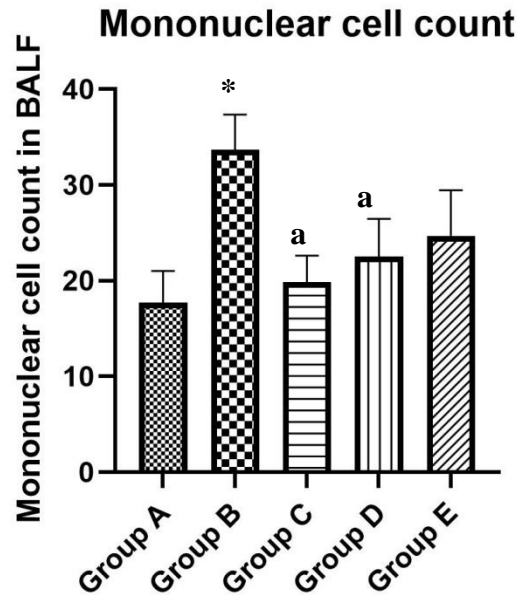


Fig. 3: Effect of tamsulosin on mononuclear cells in the BALF in rats. OVA-sensitization was accompanied by an increase in mononuclear cell count in the BALF, whereas treatment with 35 mcg/kg tamsulosin showed more reduction of these cells than the lower dose of tamsulosin 17.5 mcg/kg. Group A= normal control; Group B= negative control (OVA- sensitized airway); Group C= positive control (treated with prednisolone); Groups D and E= treated with tamsulosin (35 and 17.5 mcg/kg respectively); *= significant difference ($P < 0.05$) in comparison to group A; a= significant difference ($P < 0.05$) in comparison to group B.

Effect of Tamsulosin on Total White Blood Cell (WBC) Count/ μ l in the BALF of Rats with OVA- Sensitized Airways

The total WBC count in the BALF was significantly ($P < 0.05$) higher in the negative control group in comparison to that in the NC group (733.33 ± 63.85 versus 183.33 ± 25.77). Additionally, the total WBC count in the OVA-sensitized rats treated with prednisolone and both doses of tamsulosin (35, and 17.5 mcg/kg) were significantly ($P < 0.05$) reduced in comparison to that in the negative control (281.66 ± 33.30 , 265 ± 59.09 , and 220 ± 36.78 versus 733.33 ± 63.85), respectively, as shown in (Figure 4).

Effect of Tamsulosin on Lung Tissue Histopathology in Rats with an OVA- Induced Airway Inflammatory Model

The bronchioles and alveolar-sacs in the negative control were found to have normal epithelium by histopathological analysis, (Figure 5, A). The negative control group showed an increased buildup of inflammatory cells surrounding the bronchioles and the alveolar-sac (Figure 5, B). Prednisolone treatment resulted in a significant improvement in histological appearance, reduced inflammatory cell infiltration, and better clearance of bronchi and alveolar-sacs compared to the negative control group (Figure 5, C). Both doses of tamsulosin (35 and 17.5 mcg/kg) showed a reduction in leukocyte infiltration and buildup around the bronchial wall and alveolar sacs in addition to the overall improvement in lung histopathology; this effect was more pronounced in the higher dose of tamsulosin 35 mcg/kg, as shown in (Figure 5, D).

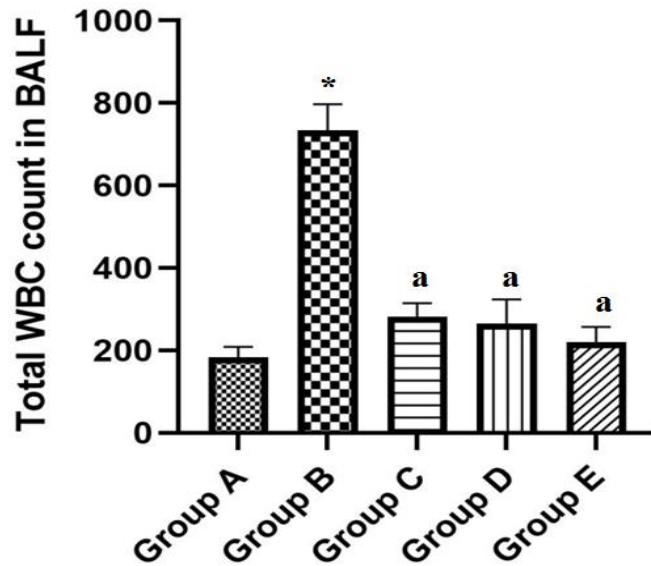


Fig. 4: Effect of tamsulosin on total WBC count in the BALF in rats. The overall number of inflammatory cells in the BALF increased after OVA sensitization. Tamsulosin treatment (35 and 17.5 mcg/kg) significantly reduced the total WBC count. Group A= normal control; Group B= negative control (OVA- sensitized airway); Group C= positive control (treated with prednisolone); Groups D and E= treated with tamsulosin (35 and 17.5 mcg/kg respectively); *= significant difference ($P<0.05$) in comparison to group A; a= significant difference ($P<0.05$) in comparison to group B.

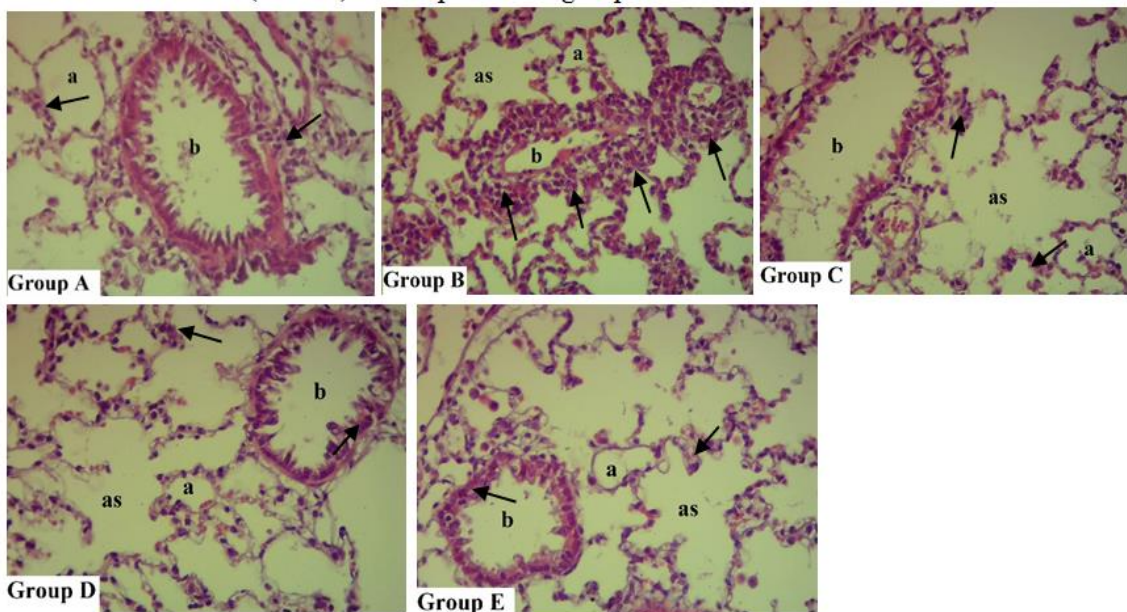


Fig. 5: Effect of tamsulosin on lung tissue histopathology in rats. OVA-sensitization was associated with an increased accumulation of inflammatory cells surrounding the bronchioles and the alveolar-sacs and the accumulation was reduced in the OVA-sensitized rats under tamsulosin treatment, demonstrating the anti-inflammatory effect of tamsulosin. Photos of lung tissue under the light microscope - X40, H&E stain. The black arrows pointing to the inflammatory cells; b, bronchi; a, alveoli; as, alveolar sac, Group A= normal control; Group B= negative control (OVA- sensitized airway); Group C= positive control (treated with prednisolone); Groups D and E= treated with tamsulosin (35 and 17.5 mcg/kg respectively).

Discussion

Corticosteroids constitute the foundation of airway-inflammation treatment due to their substantial anti-inflammatory and immunosuppressive effects. These medications have a broad list of potentially harmful side effects, particularly when used over an extended period of time³⁵. The corticosteroid prednisolone has historically been prescribed as an anti-inflammatory medication³⁶. In this study, the possible anti-inflammatory effects of tamsulosin were investigated in comparison to that of prednisolone in rats with airway-induced inflammation. The disease model was induced in rats through OVA-sensitization to mimic chronic airway inflammation³⁷. All animals in the positive control showed inflammatory effects caused by the infiltration of leukocytes into lung tissue and BALF. The foreign antigen (OVA) and the adjuvant (aluminum hydroxide) both stimulate the adaptive immune response which ultimately leads to an airway inflammation that is characterized by the infiltration of monocytes, lymphocytes, and especially eosinophils³⁸. The sensitized airway model was previously used in other studies to induce respiratory inflammation in rats by using OVA^{33&39}.

In this work, the effect of both doses of tamsulosin (35 and 17.5 mcg/kg/d) to alleviate eosinophil infiltration in the BALF was similar to that of prednisolone, indicating the ability of tamsulosin to prevent eosinophil activation, which led to the reduction of eosinophil-induced inflammation and remodeling of the airways.

Activated neutrophil cells in the airways have been linked to tissue damage and remodeling throughout the inflammatory process, and maybe a factor in organ damage and increased exacerbation frequency¹³. A marked decrease in neutrophils was linked to the leukocyte inhibitory action of both doses of tamsulosin (35 and 17.5 mcg/kg/d). In addition, the neutrophil-reducing effects of tamsulosin were similar to that of prednisolone and the NC group, leading to a reduction in the severity of airway inflammation. Another study conducted by Duan *et al.* agrees with these findings by showing that tamsulosin administration was linked to the reduction of neutrophils⁴⁰. Moreover, these results agree with previous research that showed early α_1 -AR inhibition

could reverse neutrophils accumulation in the lungs⁴¹.

Macrophages are inflammatory mononuclear cells that are more prevalent in the airways, alveolar regions, and BALF. They are associated with inflammatory processes and the breakdown of the alveolar wall during airway inflammation¹³. In this study, similar to prednisolone, tamsulosin 35 mcg/kg/d was found to reduce the accumulation of mononuclear cells in the BALF. Based on these results, tamsulosin has shown an inhibitory effect on airway inflammation, which may be related to the reduction of mononuclear cells and the associated decrease in inflammatory cytokines released by these cells.

Tamsulosin in both doses (35 and 17.5 mcg/kg/d) showed a reduction in the total WBC count/ μ l in the BALF. This data was in agreement with a previous study which confirmed that the WBC count for patients decreased significantly after treatment with tamsulosin⁴². Inflammation is a cardinal feature of chronic airway diseases which ultimately leads to impaired pulmonary function and lung remodeling. Therefore, tamsulosin was shown to have an anti-inflammatory effect by protecting against WBC infiltration into the lung tissue.

No other research has investigated the anti-inflammatory mechanism of tamsulosin through the reduction of airway inflammatory cells. However, based on other studies, we suggest that tamsulosin might act by decreasing the activation of nuclear factor Kappa-B (NF κ B). Several previous studies have shown that airway inflammation is mediated by the activation of NF κ B by inflammatory mediators. Following that, gene transcription and expression of numerous inflammatory mediators are induced, ultimately stimulating T-helper 2 cell responses and leukocyte infiltration^{43&44}. Additionally, NF κ B plays a crucial role in controlling immunological response, cell differentiation, and proliferation⁴⁵. In previous studies, the ability of tamsulosin to decrease the activation of NF κ B was successfully demonstrated^{24,46}. Similarly, prednisolone can interfere with the transcription factor (NF κ B) by inhibiting its activity, thus suppressing the inflammatory response⁴⁷. Therefore, tamsulosin might have a

similar mechanism to that of steroids in regulating inflammation on a molecular basis.

The histopathological examination of lung tissue in rats treated with prednisolone demonstrated potent inhibition of inflammatory cell accumulation in the alveoli and bronchial walls. This finding was in line with previous animal research showing that the inflammatory regions induced by OVA sensitization were efficiently decreased following treatment with prednisolone⁴⁸.

The lung tissue appeared to be protected against type-2 inflammatory events in the tamsulosin-treated groups, which resulted in improved pathological alterations in the lung tissue and caused a decreased agglomerate of inflammatory cells inside the interstitial tissue.

Conclusion

The incidence of asthma has grown significantly over the past few decades. Today, corticosteroids are still thought to be the most effective asthma medication⁴⁹. Corticosteroids, however, are known to cause a number of side effects³⁵. In order to control asthma, it is vital to find new therapy choices that are both safe and effective. This study marks the first time an α_{1A} -AR has been examined in two different doses to determine its ability to prevent induced pulmonary inflammation. The results of this current study demonstrated that tamsulosin protects against OVA-induced airway inflammation. Therefore, tamsulosin may provide hope as a possible preventative medication for asthma and COPD.

Conflict of interest

There was no conflict of interest stated by the authors of this paper.

Ethical Approval

Animal experiments were conducted within the ethical framework of the European Union Directive (86/609/EEC) of 24 November 1986 and approved by the local ethical committee board at the College of Pharmacy, Al-Basrah University

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نشرة العلوم الصيدلانية جامعة أسيوط



دراسة تأثير علاج التامسولوسين كمضاد للالتهابات في نموذج التهاب مجرى الهواء عند الجرذان من خلال تقييم الخلايا الالتهابية والتشريح النسيجي للرئة

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الأهداف: دراسة تأثير التامسولوسين على الخلايا الالتهابية في الجهاز التنفسي ويشمل تعداد الخلايا الحمضية، الخلايا العدلة، الخلايا أحادية النواة والتعداد الكلي لخلايا الدم البيضاء في سائل غسل القصبات الهوائية بالإضافة الى دراسة تشريح انسجة الرئة في نموذج التهاب مجرى الهواء عند الجرذان .

الطريقة: تم استخدام ثلاثون جرذا من الذكور، وزنها 150-250 جم قسمت إلى 5 مجموعات ، كل مجموعة تحتوي على 6 جرذان. المجموعة الاولى: مجموعة السيطرة ، أعطيت الفئران الغذاء و الماء لمدة 14 يوماً. المجموعة الثانية: تم تعريضها لتحسس مجرى الهواء فقط. المجموعة الثالثة: اعطيت علاج البريدنيزولون (4,12 مجم / كجم) عن طريق الفم بالإضافة إلى تحسس مجرى الهواء. المجموعة الرابعة: اعطيت علاج التامسولوسين (35 ميكروجرام / كجم) عن طريق الفم بالإضافة إلى تحسس مجرى الهواء. المجموعة الخامسة: اعطيت علاج التامسولوسين (17,5 ميكروجرام / كجم) عن طريق الفم بالإضافة إلى تحسس مجرى الهواء.

النتائج: انخفاض معنوي ($P\text{-value} < 0.05$) في التعداد الكلي لخلايا الدم البيضاء، الخلايا الحمضية، والخلايا العدلة للمجموعتين الرابعة والخامسة عند اعطاء علاج التامسولوسين. لكن المجموعة الرابعة فقط أظهرت انخفاضاً معنوياً ($P\text{-value} < 0.05$) في تعداد الخلايا وحيدة النواة. بالإضافة الى عكس التغييرات الالتهابية في أنسجة الرئة بعد استحداث التهاب مجرى الهواء عند الجرذان.

الاستنتاج: التامسولوسين يمكن ان يستخدم كعلاج مضاد لالتهابات الجهاز التنفسي.