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Aspirine Exacerbated Respiratory Disease and Relationship with the Salicylates Intake

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Abstract

Aspirin exacerbated respiratory disease is a condition that is often treated by the otolaryngologist, allergist and pulmonologist as a whole; it includes the definition of the disease, the clinical picture, pathophysiology, the relationship that has been found with the consumption of salicylates and recent studies on the subject.

Keywords: AERD; Asthma; Salicylate; Rhinosinusitis; Polyposis; Leukotriene

Abbreviations: AERD- Aspirin Exacerbated Respiratory Disease; CysLTs- Cysteinyl leukotriene receptor 1; LTEu4

Introduction

Aspirin Exacerbated Respiratory Disease [AERD] is a chronic inflammatory disease that affects the lower and upper airway, and is characterized by the presence of asthma, eosinophilic rhinosinusitis, nasal polyposis and hypersensitivity to aspirin or any other cyclooxygenase inhibitor-1 [1,2]. The known pathophysiology is due to the overproduction of cysteinyl-leukotrienes. These metabolites can produce edema, bronchoconstriction and mucosal hypersecretion [1,3,4].

The syndrome begins between the third and fourth decade of life, with severe nasal congestion that progresses to eosinophilic rhinosinusitis and recurrent nasal polyposis, meanwhile the symptoms of the lower respiratory tract begin and an asthma diagnosis is made 2 or 3 years later [2,3].

The typical post-intake reaction of nonsteroidal anti-inflammatory drugs appears within 30 to 120 minutes after taking; bronchospasm, rhinorrhea, nasal congestion and conjunctival irritation. The reactions vary in severity, from an isolated rhinitis to an anaphylactoid reaction with hypotension and loss of consciousness [2,5].

The most common analgesics in inducing a respiratory reaction in the United States and Europe are 80% aspirin, 41% ibuprofen, 4% naproxen and 1% ketorolac [2]. Urinary leukotriene E_4 is a biomarker of total production and excretion of cysteinyl leukotrienes. Its elevation in urine has been correlated with the total concentrations of CysLTs [1,4]. In patients with EREA it has been seen that urinary LTE_4 is 3-5 times higher compared to aspirin-tolerant patients and increases much more after the intake of COX-1 inhibitor medications and possibly with the consumption of foods high in salicylates.

The detection of salicylic acid in serum and urine in people who do not use salicylate-based drugs suggests an exogenous origin. Since salicylic acid is widely distributed throughout the plant kingdom, its presence in humans can originate from the consumption of plant-based foods [6].

Salicylates are present in large quantities in fruits and vegetables, wines, tea, fruit juices, herbs and spices [7]. Additional oral sources of salicylates include toothpaste, mouthwash and food preservatives [8]. Increased concentrations of salicylic acid and metabolites in serum and urine have been observed following acute and sustained consumption of salicylate-rich foods. Serum concentrations are linearly related to the number of servings consumed per day [6].

Discussion

A relationship has been found between salicylate consumption and respiratory symptoms in patients with asthma and association with nasal symptoms. In a study conducted by Paterson et al., It was found that 1 hour after consuming food with salicylates, serum salicylic acid concentration increased rapidly and reached a maximum value after 1.5 h and returned to baseline levels 5 hours after consumption of food. Salicylic acid derived from ingested

food appeared in the urine when the excretion rate of salicyluric acid was at its highest point [8,9]. Lawrence et al. [10] Reported higher levels of salicylic acid in the urine of vegetarian people, probably due to the higher consumption of fruits and vegetables; however, the levels are not higher compared to patients who took 75 to 150mg of aspirin.

Due to the difficulties in the implementation of a diet of avoidance of foods rich in salicylic acid and the discrepancies in the content of salicylate between studies it is impossible to give any recommendation on dietary measures for patients. A dietary salicylate restriction should only be applied when these conditions are accompanied by a very clear history of reactions to foods rich in natural salicylate [11].

At the beginning of 2015, Sommer published a pilot study in patients with EREA where he compared a low salicylate diet vs a regular diet, evaluating clinical scales such as SNOT-22, ACQ7 asthma control questionnaire, NSSS sinus symptoms scale, Lund-Kennedy and nasal endoscopy. Finding improvement on all scales except ACQ7 when undergoing a low salicylate diet for a period of 6 weeks; However, the study has some limitations such as the sample number and the inconsistency in the levels of salicylates reported in food [12]. Subsequently, a multicenter study was carried out where the same variables were measured, finding a clinically and statistically significant difference in all scales, presenting the low salicylate diet as a novel auxiliary in the treatment of the disease [13]. From the publications, other authors mention the salicylate diet and its role in the treatment of AERD [14,15].

At the National Institute of Respiratory Diseases in Mexico we conducted a study where we included 9 patients with AERD and 8 healthy controls. During the first hospitalization they received a low salicylate diet, a baseline measurement of spirometry, rhinomanometry and measurement of LTE4u was made and these measurements were repeated two hours after breakfast, lunch and dinner. In the second hospitalization they received a diet high in salicylates, making the same measurements in the established schedules. As a result, there is an elevation of LTE4u after the intake of a diet high in salicylates. We conclude that A diet low in salicylates could have a clinical utility in a sub-group of patients with AERD and greater sensitivity to salicylates in the diet [16].

Conclusion

These initial studies set the antecedent for future studies, where more patients and more measurements can be included, to generate more evidence and possibly include in the clinical practice guidelines the recommendation of a diet without salicylates as part of the treatment.

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