Antidepressants And Asthma Treatment

G Krommydas, K Gourgoulianis, V Raftopoulos, E Kotrotsiou

Citation

G Krommydas, K Gourgoulianis, V Raftopoulos, E Kotrotsiou. *Antidepressants And Asthma Treatment*. The Internet Journal of Pulmonary Medicine. 2005 Volume 6 Number 1.

Abstract

Antidepressants may be of therapeutic value in asthma. Tricyclic antidepressants have been tested in asthma therapy in the past. The beneficial effect of amitryptiline was observed since 1965, while doxepine, a tricyclic antidepressant might actually have a bronchodilator effect($_1$). There is increasing evidence that a biological linkage may exist between asthma and depression ($_{2,3}$) . Defects in the function of the autonomic nervous system such as a-adrenergic and cholinergic hyperresponsiveness and b-adrenergic hyporesponsiveness even distal from the airways has been demonstrated in asthmatic patients ,as well as in depression($_3$). Antidepressants may have a therapeutic role in asthma by supressing proinflammatory cytokines and preventing their brain effects($_4$). They also interfere with cholinergic and serotoninergic pathways, both centrally and peripherally. Most antidepressants also induce adaptive changes in central monoaminergic neurotransmission, which itself might modulate immune reactivity ($_4$).

Although most antidepressant drugs may exert an anti-inflammatory effect in bronchi and may also cause bronchodilatation, their side effects , especially those of SSRIs, raised questions in their probable future use in treating asthma, as serotonin may actually cause bronchoconstriction, thus cancelling the beneficial effects of these drugs to airways. Serotonin induces bronchoconstriction via peripheral and central pathways resulting in increasing colinergic activity and histamine release($_5$). Antidepressants that combine anti -inflammatory and bronchodilating properties with minor side effects could turn out to be promising drugs in treating asthma. For example , tianeptine is an antidepressant drug that has been recently used with success in the treatment of asthma. Tianeptine treats depression through the enhancement of serotonin reuptake from the synaptic cleft by serotoninergic terminals. It works by a mechanism that is just the pollar opposite of selective serotonin reuptake inhibition. It has been reported that this substance provoked a dramatic disappearance of clinical symptoms and improved the pulmonary function in asthmatic patients($_5$). Future development of antidepressant drugs based on these observations may actually result in new indications for antidepressants . They could also help in understanding some common pathophysiological mechanisms existing between asthma and depression .

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Author Information

Georgios Krommydas, M.D., Ph.D.
Nursing Department, High Technological Education Institute of Larissa

K.I. Gourgoulianis
Professor of Pulmology, Pulmonary Department, Medical School, University of Thessaly

Vasilios Raftopoulos, R.N., Ph.D.
Nursing Department, High Technological Education Institute of Larissa

Evangelia Kotrotsiou, R.N.
Associate Professor, Nursing Department, High Technological Education Institute of Larissa